THESIS

FOR THE AWARD OF DEGREE OF

MASTER OF SURGERY (General Surgery)

SESSION 2020-2023

"A COMPARATIVE STUDY OF OUTCOMES OF ACUTE CHOLECYSTITIS AMONG DIABETIC AND NON DIABETICS "

DR. PRIYANKA JAISWAL

SANTOSH DEEMED TO BE UNIVERSITY
NO. 1, SANTOSH NAGAR,
GHAZIABAD-201009, NCR DELHI, INDIA

DEPARTMENT OF GENERAL SURGERY
SANTOSH MEDICAL COLLEGE & HOSPITAL
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ACKNOWLEDGEMENT

"Success is determined not by whether or not you face obstacles, but by your reaction to them. And if you look at these obstacles as a containing fence, they become your excuse for failure. If you look at them as a hurdle, each one strengthens you for the next."

- Benjamin Solomon Carson Sr.

Having completed my under-graduation around 3 years ago, I have had roller coaster ride of my life before I could attain my place in this institution for the degree like many other students, of my dreams. All my family, relatives, friends, seniors, employers and all other well-wishers, whom I have been blessed to have in large numbers, tried to convince me, either out of love or anger or frustration or reasoning, to either give up on my dream subject or compromise on it and look for alternatives. But the pleasure of this success is not only soothing over all my past failures but also immensely overwhelming for all my loved ones including me.

The road to success is never easy but once achieved all roads to the goal seems like a bed of flowers giving you fragrance of knowledge and divinity of experience.

With a heartful modesty, it is a genuine pleasure to express my deep sense of thanks and gratitude to my mentor, teacher and Guide ***Dr. Shalabh Gupta****, Dean academics and Professor Department of General Surgery. It has been my delightful pleasure to walk under the shadow of such an eminent advisor and mentor for my study. I am grateful for his unvarying support, timely counsel, meticulous inspection, scientific approach and scholarly advice and guidance throughout my post-graduation which has helped me accomplish this research. He has always been approachable, understanding, calm and reasonable to our problems and with nippy and witty solutions for the same.*

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No study can be completed without the co-operation of the patients involved and hence I express my sincere thanks and respect to all my patients from whom I have learned so much during this research. Their righteous feedbacks have been the true essence of my research.

Above all, I remember our divine GODs for giving me this opportunity, and blessing me with such empowering teachers, institution, intellect and skill in accomplishing this work as success.

Dr. Priyanka Jaiswal

(Batch 2020-2023)

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INTRODUCTION

INTRODUCTION

Acute cholecystitis is the inflammation of the gallbladder. The pathophysiologic mechanism of acute cholecystitis is cystic duct obstruction. Unresolved cystic duct occlusion distinguishes biliary colic from acute cholecystitis. The blockage in biliary colic is transient and self-limiting. The blockage does not dissolve in acute cholecystitis, and inflammation occurs, resulting in oedema and sub-serosal bleeding. The gall bladder will develop to ischemia and necrosis if the blockage is not removed. Acute cholecystitis eventually progresses to gangrenous cholecystitis and, when exacerbated by infection with a gas-forming bacterium, to acute emphysematous cholecystitis.[1]

Fever, right upper quadrant discomfort, soreness to palpation, and right quadrant guarding are symptoms of inflammatory alterations in the gall bladder wall. [1] Tenderness and the presence of the murphy sign aid in differentiating acute cholecystitis from biliary colic. Mild increases in alkaline phosphatase, bilirubin, and transaminase levels, as well as a leukocytosis, all point to acute cholecystitis. Acute cholecystitis is the most prevalent consequence of gallstone disease and a leading cause of stomach pain, particularly in middle-aged women and the elderly.[1]

Acute cholecystitis is caused by an obstruction of the cystic duct, which produces inflammation. Normally, bile is produced in the liver, travels through the bile duct, and is stored in the gallbladder. After eating certain meals, particularly spicy or oily foods, the gallbladder is prompted to empty the gallbladder's bile into the duodenum via the cystic duct and the bile duct. This procedure assists in the digestion of meals.[1]

The gallbladder not only stores bile, but it also has the ability to concentrate it. When homeostasis is upset, which may occur owing to bile stasis, supersaturation of cholesterol and lipids from the liver, disturbance in the concentration process, and cholesterol crystal nucleation, concentrated bile is vulnerable to precipitation creating stones.[2]

Acute calculous cholecystitis occurs when a stone blocks the cystic duct. It is crucial to understand that discomfort caused by a brief blockage by gallstones is known as biliary colic. If the pain does not go away after six hours, the diagnosis of biliary colic is changed to acute calculous cholecystitis. If no stone is found, the condition is known as acute acalculous cholecystitis.[2,3]

Whatever the origin of the obstruction, gallbladder wall oedema will ultimately lead to wall ischemia and gangrene. Gas-forming organisms may infect the gangrenous gallbladder, producing acute emphysematous cholecystitis; all of these disorders can rapidly become life-threatening, and rupture has a high fatality rate. Gallstones are present in around 95% of persons with acute

cholecystitis.[4] However, because it is estimated that only 20% of patients with asymptomatic stones will develop symptoms within 20 years, and because approximately 1% of patients with asymptomatic stones develop complications of their stones before the onset of symptoms, prophylactic cholecystectomy is not warranted in asymptomatic patients.

With the global incidence of diabetes mellitus (DM) increasing, greater understanding of the varied clinical spectrum of biliary problems in diabetic patients should lead to earlier diagnosis and better results.[5] In contrast to William Mayo's famous comment that "there is no innocent gallstone," new research on the natural course of cholelithiasis show that fewer than one-third of people with gallstones will ultimately become symptomatic. Although the same finding holds true for diabetic individuals, they tend to have more serious problems, have longer hospital admissions, and have a higher death rate. As a consequence, prior to the age of evidence based medicine, the logic driven inclination to propose preventative cholecystectomies in this group of patients remained a perpetuated paradigm for many generations of surgeons.[6]

Even today, there are various contentious issues surrounding the diagnosis and treatment of gallstones and acute cholecystitis in diabetes individuals with classical symptoms that have varied clinical meanings. These are due to diabetic neuropathy, decreased host response to infection, and structural tissue damage induced by long-term hyperglycemia exposure.

Cholelithiasis and Diabetes mellitus have an effect on each other's clinical course, which has motivated study in both of them over the last few decades.[7] Cholecystitis, defined as gallbladder inflammation, occurs in 90% of cases due to gallbladder stones (calculus cholecystitis), with the other 10% reflecting acalculous cholecystitis. Gallstones affect around 10%–15% of the adult Western population. In a year, between 1% and 4% of people develop symptoms. In the United States alone, almost 500,000 cholecystectomies are done each year. Gallbladder illness is becoming more common in India, and so cholecystectomies are becoming more common.[8]

Gallstones affect around 10% of the population, and cholecystectomy is the most frequent kind of major surgery in Western nations. Laparoscopic Cholecystectomy is a safe and effective way to treat cholecystitis. When compared to elective Laparoscopic Cholecystectomy, it is more likely to result in laparotomy. Although laparoscopic cholecystectomy is the conventional therapy for symptomatic gallstones, its significance in the treatment of acute cholecystitis remains unknown. As a result, there are still contraindications in this case. [9]

Even for acute cholecystitis, this approach is considered first-line therapy. Medical and non-surgical therapy approaches were also compared with emergency surgical procedures for the treatment of acute cholecystitis in studies. Acute cholecystitis, on the other hand, may be fatal. [10] Laparoscopic Cholecystectomy was first performed successfully in 1882, and small-incision (S.I.C)

Cholecystectomy was developed in 1970. The latter strategy resulted in a reduction in patient morbidity. Philip Mort established laparoscopy as a surgical technique in France in 1987. This approach eventually became the preferred way for treating gallstones. [11]

Because of several technological issues and limits, open Cholecystectomy is still extensively employed, and 5 to 15% of laparoscopic surgeries eventually end up as open Cholecystectomy operations during operations. As a consequence, operation time, expenses, and morbidity all rise. Acute cholecystitis, gangrene, gallbladder empyema, obesity, pregnancy, and prior upper abdominal surgery were once recognised as Laparoscopic Cholecystectomy contraindications. These disorders are now solely recognised as laparoscopic risk factors, and they merely complicate the surgery. These risk factors are the reasons for the conversion of a laparoscopic cholecystectomy to an open cholecystectomy. [12]

Prophylactic Cholecystectomy, on the other hand, has an indication in diabetic elderly individuals. As a result, even asymptomatic elderly diabetics may be candidates for Cholecystectomy. Diabetic patients are more prone to several surgical complications owing to their pre-existing conditions, and hence their morbidity and death rates are greater. As a result, the optimum Cholecystectomy operation for these individuals has yet to be found.

Diabetes Mellitus is the world's most prevalent endocrine illness. Due to differences in diagnostic techniques, determining the real prevalence of this illness in the global population is similarly problematic. However, according to current figures, the global prevalence of this condition is 1 to 2%.[13]

This illness has the following characteristics: metabolic problems, long-term consequences (including the eyes, kidneys, nerves, and arteries), and basal membrane lesions that may be seen with electron microscopes. Because of the prevalence of diabetes mellitus and the likelihood of operating on diabetics, the surgeon must constantly be aware of diabetic patients' concerns before, during, and after surgery. The surgeon should also be aware of the potential negative effects of surgery in these patients, as well as how to deal with them. [14]

Because microvascular and macrovascular problems and neuropathic alterations are directly proportional to the length of uncontrolled hyperglycemia, the duration of diabetes may have a role in the development of severe cases. Diabetes alters the immune system and makes the diabetic more vulnerable to infections via a variety of processes including glycosylation of complement proteins, suppression of immunoglobulin mediated opsonization of bacteria, reduction of neutrophil migration, phagocytosis, and apoptosis. As a result, strict glycemic control (between 100 and 140 mg/ml) plays a critical role in reducing healthcare associated infections and promoting rapid surgical site healing. The

current research aims to assess the clinical, paraclinical, intraoperative findings, and postoperative consequences in diabetes vs. non-diabetic individuals with acute cholecystitis.

AIMS AND OBJECTIVES

AIMS AND OBJECTIVE

The aim of this study is to compare Acute Cholecystitis between Diabetic and Non- Diabetic patients under the following -

Objective

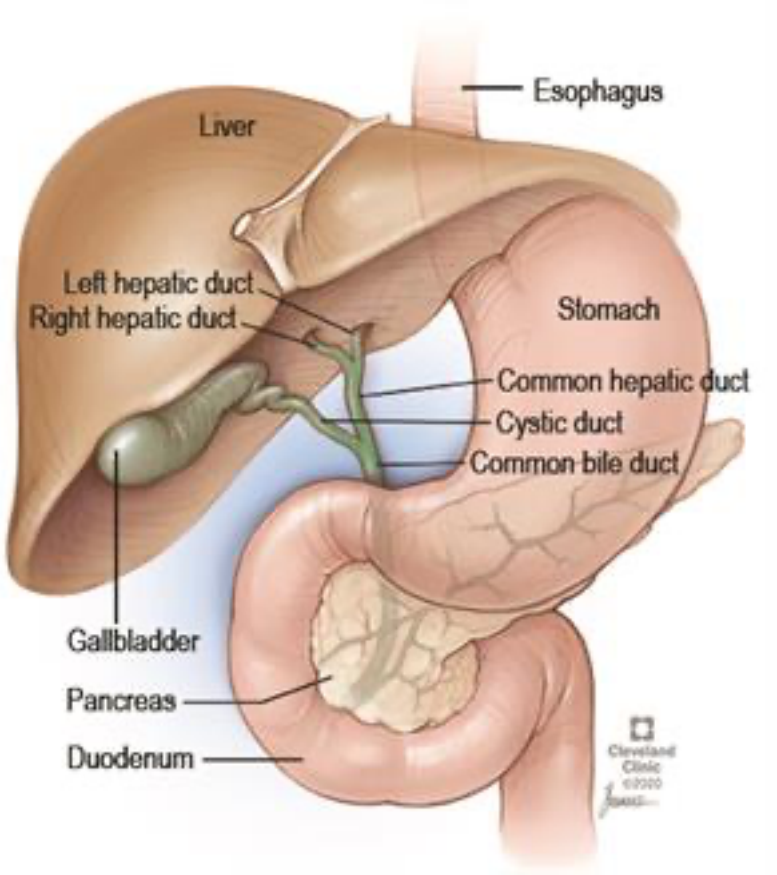
1) To compare clinical and therapeutic features among diabetic & non- diabetic patients.
2) To compare para-clinical, intraoperative & post operative findings in diabetic & non diabetic patients.

3) To diagnose acute cholecystitis in diabetic with the help of different modalities of investigations.
4) Management of acute cholecystitis in diabetic & non diabetic patients.

REVIEW OF LITERATURE

REVIEW OF LITERATURE

The term "acute cholecystitis" refers to gallbladder inflammation. The pathophysiologic mechanism of acute cholecystitis is cystic duct obstruction. Cholecystitis is best treated surgically, however it may also be managed conservatively if required. This disorder may be acute or chronic, and it can be linked with or without the presence of gallstones. It is seen in both males and women, but may be more prevalent in specific cultures. It may also exhibit some of the traditional signs and symptoms. Acute cholecystitis might be misdiagnosed as peptic ulcer disease, irritable bowel disease, or heart disease. Pancreatitis, both chronic and acute, may look like gallbladder disease. [15-17]



Epidemiology

Cholecystitis is responsible for 3–10% of all instances of stomach discomfort globally. In the United States in 2012, cholecystitis was responsible for an estimated 651,829 emergency department visits and 389,180 hospital admissions. In 2012, the mortality rate in the United States was 0.7 per 100,000 persons. Cholecystitis is more common in adults aged 50–69 years old. [18]

Acute Cholecystitis Types

They are primarily divided into two types:

1. Cholecystitis with calculi
2. Acute cholecystitis with calculi

Cholecystitis calculos aacute

Gallstones that obstruct the passage of bile account for 90% of cholecystitis cases (acute calculous cholecystitis).[19] Bile flow obstruction causes bile thickening and accumulation, resulting in an enlarged, red, and tight gallbladder.[19] The gallbladder is originally sterile, but bacteria, primarily E. coli, Klebsiella, Streptococcus, and Clostridium species, often invade it. Inflammation may migrate to the gallbladder's outer layer and nearby tissues such as the diaphragm, resulting in referred right shoulder pain.[19]

Cholecystitis acalculous

There is no stone in the biliary ducts in acalculous cholecystitis.[20] It accounts for 5–10% of all cholecystitis cases and is linked with substantial morbidity and death rates. Acalculous cholecystitis is most often observed in severely unwell patients who are hospitalised.[20] In the absence of trauma, males are more prone to develop acute cholecystitis after surgery.[21] It is caused by a variety of factors, including vasculitis, chemotherapy, significant trauma, or burns. The symptoms of acalculous cholecystitis are similar to those of calculous cholecystitis.[20] Patients with calculous cholecystitis are more prone to suffer skin yellowing (jaundice). An immobile, enlarged gallbladder is often seen on ultrasonography or computed tomography. Antibiotics are administered immediately, and a cholecystectomy is performed within 24–72 hours. [21]

Cholecystitis chronica

Chronic cholecystitis develops after many bouts of acute cholecystitis and is virtually invariably caused by gallstones. Chronic cholecystitis may be asymptomatic, manifest as a more severe case of acute cholecystitis, or result in a variety of consequences such as gangrene, perforation, or fistula development. [20,21]

Xanthogranulomatous cholecystitis (XGC) is an uncommon type of chronic cholecystitis that looks like gallbladder cancer but is not. McCoy and colleagues published the first report in the medical literature in 1976.[22] Over 90% of instances of acute cholecystitis are caused by a blockage of the cystic duct by gall stones or biliary sludge that has gotten impacted at the gall bladder's neck. The obstruction of the cystic duct raises intraluminal pressure inside the gallbladder, triggering an immediate inflammatory response along with cholesterol supersaturated bile. Gallstone trauma promotes the creation of prostaglandins I2 and E2, which mediate the inflammatory response.[23] In roughly 20% of instances, secondary bacterial infection with enteric organisms (most often Escherichia coli, Klebsiella, and Streptococcus faecalis) occurs. Biliary sludge is a combination of particulate matter and bile that has the potential to cause microlithiasis. Gall stones may occur if the sludge continues, for example, because the patient has previously had numerous pregnancies or is receiving whole parenteral nourishment. [24] Although most people with biliary sludge have no symptoms, the sludge may cause acute cholecystitis.

Symptoms and signs

The majority of persons with gallstones have no symptoms.[19] Biliary colic occurs when a gallstone becomes temporarily lodged in the cystic duct.[19] Abdominal discomfort in the right upper quadrant or epigastric area is a symptom of biliary colic. It is an episodic condition that occurs after consuming greasy or fatty meals and causes nausea and/or vomiting.[20] People who have cholecystitis often experience biliary colic symptoms before developing cholecystitis. Cholecystitis causes significant and ongoing discomfort. Nausea is prevalent, and 75 percent of persons with cholecystitis vomit.[21] Aside from stomach discomfort, right shoulder ache is possible. [20]

An inflamed gallbladder is nearly always sensitive to the touch and palpable (25-50 percent of the time) at the midclavicular right lower rib border on physical examination.[20] In addition, fever is prevalent. Touching a gallbladder with cholecystitis is nearly always painful.[20] Because of the inflammation, the size of the gallbladder may be felt from the outside of the body in 25–50% of persons with cholecystitis.[20] Severe discomfort is frequently caused by deep inhalation leading to the termination of the breath while pushing on the right upper quadrant of the abdomen (Murphy's sign). Jaundice (yellowing of the skin) might develop, although it is usually moderate. Severe jaundice indicates the presence of another source of symptoms, such as choledocholithiasis. [21] People who are elderly, have diabetes, have a chronic condition, or are immunocompromised may have nonspecific symptoms that may not involve temperature or localised discomfort. [25]

Complications

If cholecystitis is not discovered and treated promptly, it may lead to a variety of issues. High temperature, shock, and jaundice are all symptoms of complications. Among the complications are the following:

- Gangrene
- Rupture of the gallbladder
- Empyema
- Formation of a fistula with gallstone ileus
- Sinuses Rokitansky-Aschoff
- Gallbladder rupture and gangrene

Cholecystitis causes the gallbladder to swell and become hard. Distension may reduce blood supply to the gallbladder, resulting in tissue death and, ultimately, gangrene. When tissue dies, the gallbladder is at a much higher risk of rupture (perforation), which may cause severe discomfort. Rupture may also happen in chronic cholecystitis. [20] Rupture is an uncommon but significant consequence that may result in an abscess or peritonitis. [21] A massive rupture of the gallbladder has a 30% mortality rate. [20]

Empyema

Untreated cholecystitis may result in exacerbated inflammation and infected bile, which can develop to empyema, an accumulation of pus within the gallbladder.[20] Empyema symptoms are comparable to those of simple cholecystitis, but more severe: high fever, severe abdominal discomfort, and a more significantly increased white blood count. [20]

Formation of a fistula with gallstone ileus

Cholecystitis inflammation may cause adhesions between the gallbladder and other regions of the gastrointestinal system, most often the duodenum. These adhesions might result in the creation of fistulas, which are direct connections between the gallbladder and the gastrointestinal system. Gallstones may travel from the gallbladder to the intestines through these direct connections. Gallstones may get lodged in the digestive system, most often near the junction of the small and large intestines (ileocecal valve). When a gallstone becomes stuck, it may cause an intestinal blockage known as gallstone ileus, which causes stomach discomfort, vomiting, constipation, and distension. [20]

ANATOMY

Understanding the architecture of the gallbladder and the extrahepatic biliary system is critical for making an accurate diagnosis and managing gallbladder illness. This characteristic is highlighted by the organ's proximity to a number of important arterial structures as well as other major viscera of both the gastrointestinal and genitourinary tracts. Biliary abnormalities are prevalent, and failing to diagnose such a congenital issue may result in considerable perioperative morbidity and death. The gall bladder is a hollow sac-like structure that rests in a fossa on the underside of the liver. Its location determines the anatomic border between the liver's right and left lobes, as well as the division of the arterial and ductal systems. The gallbladder is connected to the liver by loose areolar tissue that is densely packed with tiny blood arteries and lymphatics. A peritoneum covers the extrahepatic region of the gall bladder. The gall bladder is usually located near the duodenum, pylorus, hepatic flexure of the right colon, and right kidney.[26]

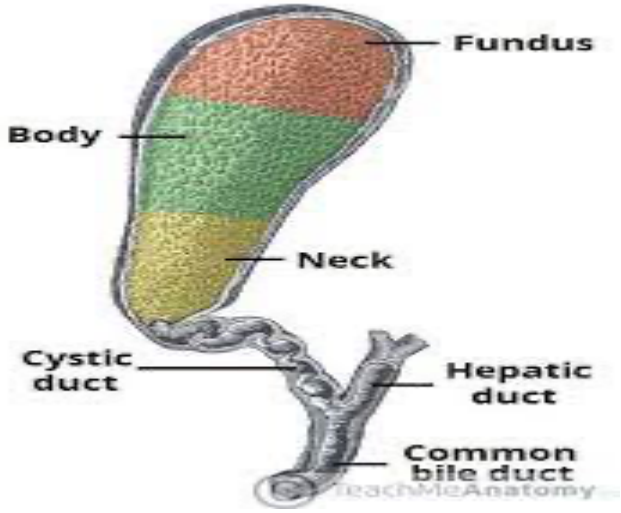
The gall bladder is classified as follows:

1. Fundus: The fundus is the rounded blind section of the gall bladder that extends for a short distance beyond the liver margin, and it is the least vascularized, making it more prone to ischemic alterations, including perforation.

2. Body: This is the bulk of the gall bladder and comes into touch with the liver, duodenum, and hepatic flexure of the colon.

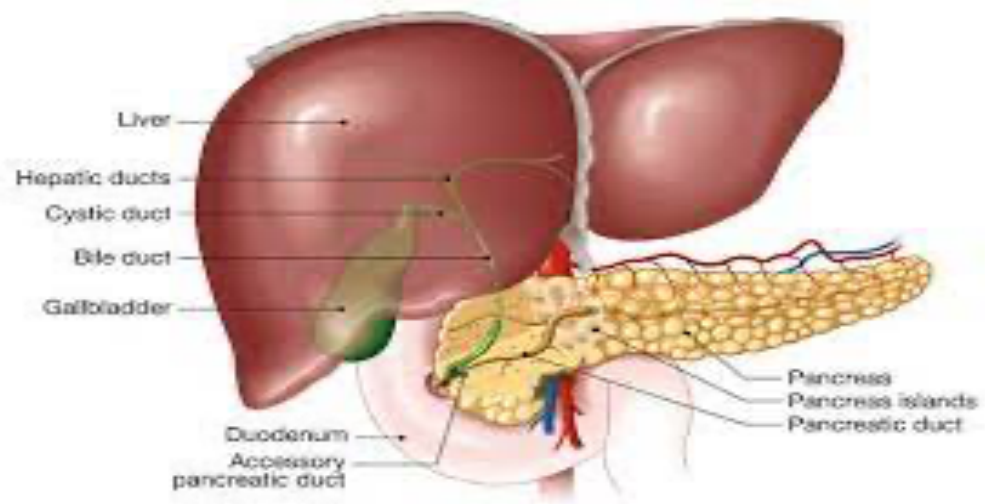
3. Infundibulum: A tiny bulbous diverticulum that usually lies on the inferior side of the gallbladder. Because stones may get lodged in the infundibulum and clog the cystic duct, this anatomic region is therapeutically relevant.

4. Neck: The thin section of the gallbladder between the body and the cystic duct region.



The Duct Cystic:

This links the gallbladder to the common bile duct, mostly in the supraduodenum (80 percent). This is typically 3 cm length with a lumen of l-3mm. The mucosa of the cystic duct is structured in spiral folds known as 'HEISTER' valves, and its wall is encircled by a sphincteric structure known as 'LUTKINS' sphincter. The spiral folds of mucosa in the cystic duct are important from a surgical standpoint because they may obstruct the passage of an instrument and also halt the transit of gallstones.



LYMPHATICS AND INNERVATION / BLOOD SUPPLY:

Arterial provision: The cystic artery, which is usually a single branch of the right hepatic artery, supplies the gall bladder. The cystic artery may also be formed by the left hepatic, common hepatic, gastroduodenal, or superior mesenteric arteries. The cystic artery is normally parallel and medial to the cystic duct, however its path might vary depending on its origin. Before accessing the gallbladder, the cystic artery separates into superficial and deep branches. The most serious abnormalities occur when the hepatic artery follows a convoluted path in front of the cystic duct's origin, or when the right hepatic artery is tortuous and the cystic artery is short. This tortuosity is often referred to as the "CATERPILLAR TURN" or "MOYNIHAN'S HUMP." This variance is the source of many complications during a difficult cholecystectomy, including inflammation in the cystic duct area and accidental injury to the right hepatic artery in the most difficult to regulate laparoscopically.

Veins:

The gallbladder's hepatic surface is emptied by many tiny veins that penetrate through the gall bladder bed and split up into capillaries inside the liver material. They do not form a single cystic vein, but rather a network of tiny veins that enter the liver alongside ascending veins from the common bile duct.

Lymphatics:

The gallbladder's lymphatic veins (subserosal and submucosal) drain into the cystic lymph node of "LUND" (the sentinel lymph node), which is located in the fork formed by the confluence of the cystic and common hepatic ducts. This lymph node's efferent veins connect to the liver's hilum and the celiac lymph nodes.

Innervation:

Both sympathetic and parasympathetic nerves supply the gall bladder. The celiac ganglion is where the parasympathetic nerves branch out from the vagus nerve. Sympathetic supply originates from T8 and T9 levels, where postganglionic fibres feed the gall bladder via the hepatic artery.

KEY LAND MARKERS:

The calot triangle is created by the cystic duct, the common hepatic duct, and the undersurface of the liver. The cystic artery and the cystic lymph node of rear are usually found in this location, and the cystic artery's path is commonly behind the hepatic duct.

Acute cholecystitis and pathological correlation:

Gall stones are classified into two types:

1. Gallstones caused by cholesterol predominate in Western nations, accounting for about 70% of all cases.
2. Pigment / brown stones - account for the remaining about 30%, primarily in the United Kingdom and South Asian nations such as India.

Cholesterol gallstone pathogenesis:

Gall stone production occurs in three phases.

1. Supersaturation of cholesterol
2. Nucleation
3. Stone development

Supersaturation of cholesterol:

Cholesterol, which is insoluble in water, is retained in solution by the detergent activities of bile salts and phospholipids, with which it forms a micelle. Cholesterol crystals occur in bile containing cholesterol stones because there is an excess of cholesterol compared to bile salts and phospholipids. This kind of bile is known as 'supersaturated' or 'Lithogenic.' The most significant process in the generation of lithogenic bile is increased biliary secretion of cholesterol in conditions such as:

- Obesity
- Diets high in calories
- Clofibrate treatment
- Increased HMG – COA reductase activity

Stone development and the kinetic balance of nucleating and anti-nucleating factors: Bile includes chemicals that either prevent (A lipoprotein Al) or enhance (Mucin) cholesterol crystal formation. Thus, the balance of these two kinetic parameters influences the formation of cholesterol stones. Anionic polypeptide fragment (AFP) and Immunoglobulin A are two more proteins that prevent gallstone development. It is also believed that a reduction in the absolute or relative quantity of APF promotes the nucleation and development of cholesterol monohydrate and pigment crystals. It should be noted that the gall bladder also plays an essential part in the production of gall stones (both cholesterol and pigment stones). It is clear that this organ is responsible for 85-90 percent of stone formation rather than the bile ducts.

The gall bladder may alter the physiochemical composition of bile, favouring nucleation and crystal growth through abnormal absorption / secretion, defective surface PH, stasis caused by impaired gall bladder emptying and bile stagnation, or by providing essential nucleating factors such as mucin, desquaminated cells, bacteria, and refluxed intestinal contents.

Gallstones and gall bladder mobility:

Obviously, bile crystallisation and crystal aggregation are time-consuming processes. As a result, stone development necessitates that bile stay in the gall bladder for a period of time, and it may be hypothesised that the gall bladder of gall stone formers will be less active in expelling its contents. So there is evidence that decreased gall bladder emptying is a significant pathogenic component in the production of cholesterol stones.

Pigment stone pathogenesis:

The pathophysiology of pigment stones is far less well understood.[27] These stones are often encountered in people suffering from haemolytic diseases or cirrhosis. The absence of germs in the majority of patients with black stones suggests that other causes are at work. The increased frequency of pigment gall stones in individuals with haemolytic disease presumably stems from excess loads of bilirubin being presented to liver for secretion. One probable reason for the increased occurrence of pigment stones in cirrhotic patients is hypersplenism, which causes haemolysis and impairs bilirubin conjugation, resulting in pigment stones.

Acalculous Cholecystitis and Pathological Correlation:

It is a rare but possibly fatal consequence of gallbladder illness. The actual aetiology and pathophysiology of this illness are unknown. The estimated incidence is about 3%. It has been documented in critically sick patients in intensive care units, notably in patients with MOF, after severe trauma or burns, and as a post-operative complication of any major treatment. The clinical situations in which acalculous cholecystitis occurs have revealed vital information about its probable pathophysiology. [28]

Bile stasis inside the gallbladder has long been thought to have a role in the development of this condition. Biliary spasm may develop as a consequence of ampullary spasm caused by opioids, reduced gall bladder emptying during extended fasting, cystic duct blockage caused by enema, and so on. Furthermore, changed viscosity due to dehydration and numerous transfusions may have a role. The release of particular biochemical mediators of inflammatory response sets off a chain of actions.

Ischemia may have a role, however since the cystic artery is an end artery with little collateral circulation, it may lead to organ ischemia and gangrene. The diagnosis of acalculous cholecystitis poses a significant barrier to the doctor since the patient is often in an intensive care unit. The key to the diagnosis is early ultrasonography and a strong clinical suspicion based on the clinical context. All patients need an emergency cholecystectomy, however if the patient's overall health is really bad, a temporary Cholecystostomy may be performed to tide them over until the crisis passes.

INVESTIGATION

Over the last 15-20 years, breakthroughs in diagnostic radiography and interventional endoscopy have allowed for the accurate imaging and location of gall bladder disease, as well as the type of it. Particular tests have been developed to address specific questions.

Radiography of the abdomen:

A simple x-ray of the abdomen is of little use in evaluating individuals with gall stones. Supine and upright abdominal films can help rule out other causes of abdominal pain, such as a perforated viscus or a bowel obstruction. The presence of considerable quantities of calcium inside gallstones, which occurs in only 15-20% of individuals, causes stones to show on plain x-rays as distinct objects positioned in the right upper quadrant. It is vital to note that the majority of gallstone patients will have normal abdominal x-rays. Here are a few such scenarios in which unique radiography findings may indicate morbidity. These include the presence of air inside the biliary and outer architecture in individuals with cholecystoenteric fistulas, as well as air bubbles in the gall bladder wall in patients with emphysematous cholecystitis.

a. Cholecystography orally:

Graham and Cole developed a reasonably simple test for identifying gall stones in 1924. This test may allow for the visualisation of gallstones inside the gallbladder; the essential function examined is the gallbladder's absorptive capacity.

b. Abdominal Ultrasound:

The recording of high frequency sound reflected off interfaces with different acoustic impedances serves as the foundation for this test. This has a sensitivity rate of 98% and a specificity rate of 95%.

Ultrasonograms may be used to analyse a variety of factors, including:

• Gallbladder volume

• Gallbladder capacity

• Ejection percentage

• Wall thicknesses

• Calculate the thickness

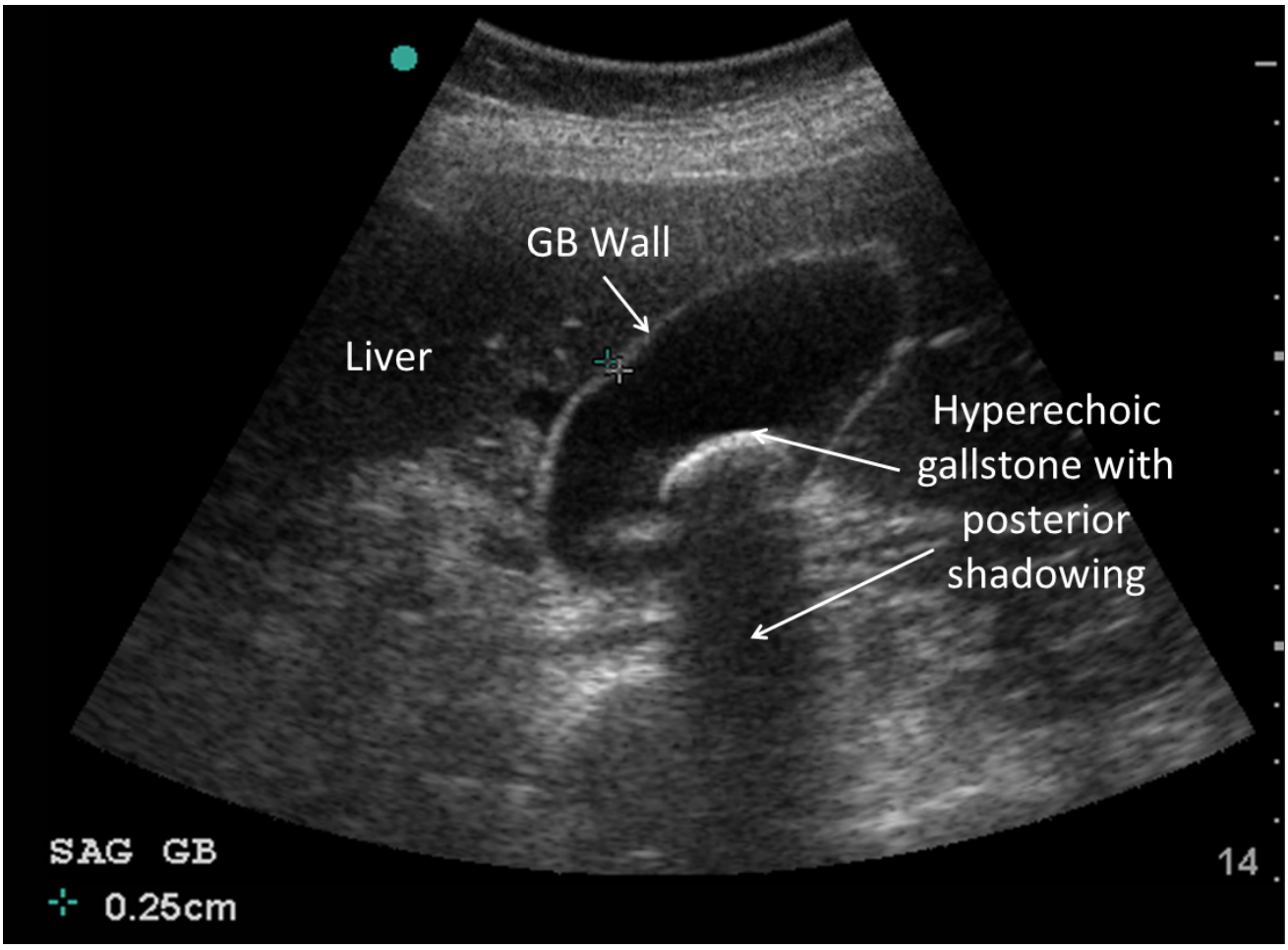
• The number of calculations

• CBD Diameter

• CBD Calculi

• Other gastrointestinal diseases

Cholelithiasis is often seen as an echogenic centre with distal acoustic shadowing that moves with position shift.



c. Bilateral scintigraphy:

The liver, gallbladder, and extrahepatic bilary tract have been imaged via intravenous injection of one of a series of technitium 99 nun tagged Imminodiacetic acid radio isotopes. The clearance of the isotope from the blood by hepatocytes and its excretion into the bile ducts is the foundation for this test. This test offers particular information on the cystic duct's patency. It is sensitive in identifying individuals with acute cholecystitis in this situation. This is a functional test, as opposed to USG, which is an anatomic exam. In reality, these two complement each other.

The radio medicines utilised are as follows: HIDA stands for ethyl hydroxy imminodiacetic acid. P1PIDA is an abbreviation for para - isopropyl acitanilido - immino - diacetic acid. An intravenous injection of 200mbq is administered, and serial analogue pictures are produced. A negative scan demonstrates passage from the liver via the biliary tree into the gallbladder and small bowel. In uncertain circumstances, further projection (for example, left anterior oblique) may be acquired. Failure to show any section of the biliary tree on scintigraphy implies hepatic dysfunction or biliary blockage. If the gall bladder is only visible after 2 hours or is not visible at all after 24 hours, the scintigraphy is deemed 'positive,' suggesting the likelihood of acute cholecystitis.

HIDA Scan vs. Ultrasound

Although ultrasound falls short in terms of specificity, it provides more information than HIDA scanning in evaluating right upper quadrant pain because it can detect other pathologies. Furthermore, it can provide pre-operative evaluation to predict technical difficulties and complications in laparoscopic cholecystectomy.

d. Computed Tomography (CT):

Unlike ultrasonography, CT scans use ionising radiation to produce pictures. This test is not especially sensitive for gall stones or inflammatory gall bladder disease, but it does offer a lot of information on the kind, size, and location of biliary dilatation, as well as masses in and around the biliary system and pancreas. In general, this test provides more useful information than ultrasound when evaluating jaundiced patients. Patient exposure to ionising radiation and expense are two limiting considerations.

Magnetic resonance imaging (MRI):

Although the pictures produced by this diagnostic test may look similar to CT scans, they are the consequence of the varying magnetic properties of the tissues. For this test, a contrast substance

called "gadolinium" is commonly employed. The magnetic properties of tissues are affected by this IV-injected substance. Its relevance in the assessment of patients with hepatobiliary illness is still unknown.

ACUTE CHOLECYTITIS MANAGEMENT MODALITIES

A. CHOLECYSTOSTOMY

Patients who are critically ill and frequently present with multisystem disease have an increased morbidity and mortality during acute cholecystitis. Cholecystostomy is classified into two categories.

1. Cholecystostomy surgery
2. Cholecystostomy guided by ultrasonography.

Cholecystostomy surgery: For select high-risk populations, surgical cholecystostomy has been proposed.[29] Patients suffering from sepsis and failing organs, Critical cardiac failure

Cholecystostomy via the skin

Hawkins tiny needle approach is used to accomplish percutaneous transhepatic cholecystostomy, which involves inserting accordion catheters into the gallbladder. Clinical criteria for improvement included quick pain defervescence resolution, reduced WBC, and early improvement in the liver function profile.[30]

B. CHOLECYSTECTOMY

1. a. Emergency cholecystectomy
   b. Postponed cholecystectomy
2. Open cholecystectomy
3. Cholecystectomy through laparoscopy
4. Cholecystectomy with a mini-lap
5. Cholecystectomy with little invasiveness

The gold standard therapy for acute cholecystitis is cholecystectomy.

Cholecystectomy in an emergency

If the patient's health worsens over the next 24 to 48 hours, an emergency cholecystectomy is performed.[31]

Cholecystectomy performed early With the exception of emergency instances, patients may be scheduled for surgery on the next available list.

Mellitus Diabetes

It is a set of metabolic illnesses characterised by persistently elevated blood sugar levels. Numerous studies have shown that diabetes people have a higher morbidity and fatality rate from acute cholecystitis. Diabetics are more prone to have advanced inflammatory abnormalities in their gallbladder. Diabetics might also anticipate a greater likelihood of gangrene, postoperative difficulties with wound healing and infection, and a nonsignificant increased risk of perforation.

TEST CRITERIA	PREDIABETES	OVERT DIABETES MELLITUS
HbA1c	5.7% to 6.4%	≥ 6.5%
Fasting plasma glucose test (mg/dL)	100 to 125	≥ 126
Plasma glucose after 75 g oral glucose tolerance test	140 to 199	2 hours: ≥ 200
Random plasma glucose test with symptoms of hyperglycemia (mg/dL)	Not applicable	≥ 200

Some of the studies in this relation are:

1. Ransohoff, D.F. et al (1987)[32] evaluated the course of acute cholecystitis in individuals . It was discovered that mortality occurred in 3 of 46 diabetic individuals and 7 of 263 non-diabetic patients. Diabetes patients had a 2.2 age-adjusted relative risk of mortality compared to nondiabetic individuals. Only one of the three diabetic patients who died had been diagnosed with diabetes during the previous five years. Individuals with elevated blood urea nitrogen levels (> 20 mg/dL) had a higher death risk when compared to patients with normal values. These findings show that the advice for preventative cholecystectomy in diabetic individuals with silent gallstones should be reconsidered.
2. Hickman M.S. et al (1988)[33] identified acute cholecystitis in diabetic individuals. This research comprised 72 diabetics who needed an emergency procedure for acute cholecystitis. Patients were matched for age, gender, and date of surgery with nondiabetic controls. A review of the patient data found no statistically significant differences in hospital stay or the severity of

surgical and pathologic findings. Diabetics, on the other hand, had much higher morbidity (38.9%) than nondiabetics (20.8 percent). Furthermore, diabetic infection-related problems occurred almost three times as often as controls (19.4 percent vs 6.9 percent). Diabetics (4.2 percent) were the only ones to die as a direct consequence of the effects of sepsis. These data imply that acute cholecystitis in diabetics is linked with a greater frequency of infection-related sequelae, highlighting the need of prompt surgical intervention in symptomatic individuals.

3. Landau O et al (1992)[34] investigated the risk of acute cholecystitis in diabetic patients, they were noticed that the diabetics are more likely to be operated on in the acute stage of their condition. The DM group showed a greater risk of septic bile, gangrenous alterations and perforations of the gallbladder wall. The DM group had a higher morbidity rate and a slightly higher death rate. The degree of increased surgical risk does not in our opinion warrant suggesting cholecystectomy in diabetic individuals with asymptomatic gallstones. Early surgery however, is strongly suggested in diabetics with symptomatic gallstones and acute cholecystitis.
4. Aucott J.N. et al. (1993)[35] investigated the therapy of gallstones in diabetic patients. Autopsy results and uncontrolled studies have demonstrated a greater incidence of cholelithiasis in diabetics, and early reports revealed drastically increased perioperative morbidity and mortality for treatment of diabetics with acute cholecystitis. As some experts have suggested preventative cholecystectomy for diabetes individuals with asymptomatic gallstones, this is in contrast to advice for nondiabetics. Recent studies have shown equal rates of operational morbidity and death for biliary surgery in diabetics and the general population. Recent research has called into question whether diabetes is a separate risk factor for gallstone development. Decision analyses utilising these new data have demonstrated that preventative cholecystectomy is not of evident benefit and should not be regularly suggested for diabetics with asymptomatic gallstones. they were determined that the asymptomatic individuals with diabetes do not benefit from screening for gall-stones and that cholecystectomy should only be done in situations of symptomatic cholelithiasis, as is the case in the general population.
5. Ardakani J. et al. (2001)[36] investigated the complications of cholecystectomy in diabetic and non-diabetic patients. it was revealed that the 23.5 percent patients of diabetes and 74 percent patients of non diabetics had a greater stages of cholecystitis. Postoperative problems were reported in 38.4 percent instances - of diabetes and 10.5 percent cases of nondiabetic groups. It appears that greater occurrence of surgical complications in diabetes patients is owing to more advanced stages of their, illness at the time of diagnosis.
6. Bedirli A. et al. (2004)[37] investigate if diabetes is a risk factor in patients undergoing laparoscopic cholecystectomy for symptomatic gallbladder stones. 862 patients were involved in this

investigation. There were 21 percent diabetes and 79 percent nondiabetic individuals. They discovered that there was no significant change in leukocyte count, bilirubin, or amylase levels between diabetes and nondiabetic subjects. Diabetic individuals had considerably greater incidence of surgical and postoperative complications. Conversion to open surgery was necessary in 2.8 percent of control patients and 7.1 percent of study patients. The operation time and duration of hospital stay did not vary substantially between the two groups. They were determined to have the same symptoms and laboratory results; nevertheless, laparoscopic cholecystectomy in diabetes individuals is linked with greater morbidity and a higher conversion rate than in nondiabetic patients.

7. Ziaee SA et al (2005)[38] investigated the risk variables related with gallstone morbidity. In this investigation, 116 individuals out of 669 were reported to have problems. Complications included 68 adhesions, 26 gangrenes of the gallbladder, 14 pancreatitis, 20 hydropses, and 5 perforations. In univariate analysis, age, diabetes, hypertension, and ischemic heart disease were substantially related with increased risk of complications. Only diabetes produced a substantial increase in complications in multivariate analysis, with an odds ratio (OR) of 6.1. Diabetes was also linked to an increased risk of adhesion, gangrene, and pancreatitis. Although this research does not explicitly support preventative cholecystectomy, the higher morbidity in diabetics suggests that diabetic individuals with asymptomatic gallstones need further care and attention.
8. Lee S. et al. (2011)[39] investigated previously recognised risk factors for acute cholecystitis as well as various co-morbidities such as hypertension, diabetes mellitus, cardiovascular disease, cerebrovascular accident, and end-stage renal disease, the incidence of which has grown in the elderly. it was revealed that the 611 patients who had laparoscopic cholecystectomy were acute cholecystitis 42.2 percent and chronic cholecystitis 57.8 percent . Male gender, age more than 50, fever, leukocytosis, AST elevation, alkaline phosphatase elevation, and total bilirubin elevation were found as risk factors for acute cholecystitis. Diabetes mellitus and hypertension may both be risk factors for acute cholecystitis. Early therapy, that is, early examination and diagnosis after early cholecystectomy, is suggested for individuals with risk factors for acute cholecystitis before the condition evolves to an acute form of cholecystitis.
9. Karamanos E. et al (2013)[40] examined the incidence of diabetes and its influence on surgical outcomes in patients having emergent, in-patient cholecystectomy for acute cholecystitis. This research covered a total of 5,460 patients. Diabetes was diagnosed in 14.10% of these people. Diabetics died at a greater rate than non-diabetics. Preoperative perforation rates were 25.1 and 13.0 percent , respectively. The adjusted risk of cardiovascular events and renal failure was considerably greater for diabetics. Insulin administration, but not oral medicine, was related with

a substantial increase in mortality, preoperative perforation, superficial surgical site infection, septic shock, cardiovascular events, and renal insufficiency. It was found that the patients having cholecystectomy for acute cholecystitis, diabetes increases the risk of death, cardiovascular events, and renal failure. Diabetics on insulin had higher co-morbidities and worse results.

10. Bansal A et al (2014)[41] assess the prevalence and therapy of cholelithiasis. Clinical symptoms were reported according to clinical history of the patients. The ultrasound scanning of the abdomen conducted, open/laparoscopic cholecystectomy was done. A varied diet was consumed, and several gallbladder calculi of a mixed variety were discovered. Wound infection was the most prevalent consequence in the open cholecystectomy group, and the average hospital stay was much longer.
11. Hosseini S.V.S. et al. (2014)[42] investigated the postoperative complications of laparoscopic cholecystectomy in diabetic individuals. Although it was shown that diabetes patients had much more problems than non-diabetic patients, no significant difference in the frequency of complications was seen between the two groups. The mean duration of surgery on diabetes and non-diabetic patients was 57.22±7.40 and 53.27±11.19 minutes, respectively. Diabetic individuals were hospitalised for much longer periods of time. The incidence of development of symptomatic gallbladder illnesses that necessitate surgery is greater in diabetes people. This research also suggests that diabetes individuals have a greater risk of post-surgical problems than non-diabetics.
12. Liu C. et al (2015)[43] evaluated the rate of cholecystectomy in diabetic individuals with GSD. It was discovered that the incidence rates of patients who had GSD and underwent cholecystectomy in the diabetes and control groups were 18.65 percent and 17.15 percent, respectively; hence, the rate of cholecystectomy was greater in the diabetic group than in the control group. Male sex and elderly age were linked to a higher risk of cholecystectomy, regardless of diabetes condition. In GSD patients, hypertension, cholangitis, and Caroli disease were linked to an increased risk of cholecystectomy. Over an eight-year period, diabetes individuals had a greater incidence of cholecystectomy than nondiabetic patients. They were concluded that the males with GSD are more likely to have cholecystectomy than women, independent of the diabetes condition; consequently, an aggressive GSD care strategy for men must be explored for early identification of these patients before the necessity for emergency surgery.
13. Ismat U. et al(2016)[44] evaluated the incidence of surgical site infections in individuals with type II diabetes following laparoscopic cholecystectomy as compared with non-diabetic patients. Patients were randomised into two groups of 60 each, having laparoscopic cholecystectomy. Group A consisted of non-diabetic individuals, whereas Group B consisted of type II diabetes

patients. they were discovered In group A, 35 patients were over the age of 40 years. In group B, 38 patients were over the age of 40 years. Four patients in group A developed a surgical site infection. In group B, seven patients had SSIs. it was established that the presence of diabetes mellitus did not substantially impact the development of surgical site infection in individuals having laparoscopic cholecystectomy.

14. Hegazya T. O. et al. (2018)[45] compared main and secondary outcomes in individuals with uncomplicated ACC treated with early LC (within 72 h) to interval LC (after 6 weeks of conservative therapy). A total of 100 patients with uncomplicated ACC are randomly assigned to one of two groups: group A for early LC and group B for interval LC. They came to the conclusion that there is no significant difference between the two groups in terms of main and secondary outcomes. Early LC is practical and safe, and interval LC has no effect on conversion rate. In group A, there is a considerably high risk of infection, with longer hospital stay in diabetics, and in group B as well, the bile leak and rate of conversion are high, with longer hospital stay in diabetics.
15. Lacka M et al (2019)[46] conducted a review to synthesise the existing research on acute cholecystitis in diabetic individuals. An electronic search was conducted for this evaluation. They found that diabetic people are more likely to experience problems. A tailored screening and treatment strategy, as well as thorough diabetic patient preparation for an elective cholecystectomy, may improve the prognosis.
16. Mahapure KS et al (2019)[47] was to identify intraoperative challenges and complications during laparoscopic cholecystectomy in diabetic and nondiabetic individuals. 60 patients were split into two equal groups. Pre-operative parameters such as clinical presentation, duration of symptoms, and vitals were shown to be equivalent in diabetes and non-diabetic patients. A much larger proportion of diabetic individuals received open pneumoperitoneum. There was a statistically significant difference in the appearance of the gallbladder, adhesions, dissection of Calot's triangle, releasing adhesions, intra-operative bleeding, grasping of the gallbladder, removal of the gallbladder from the liver bed, specimen extraction, drain placement, and conversion. Diabetes mellitus patients had considerably longer operation times. they were determined that the diabetes mellitus is strongly connected with operating problems and longer surgical time during laparoscopic cholecystectomy.
17. Escartn A. et al (2019)[48] studied the characteristics, therapy, and outcomes of acute cholecystitis in patients aged 80 years. 348 patients were separated into two groups: those younger (Group A) than the median age and those older (Group B) (85.4 years). Although the two groups' demographic and clinical features were comparable, the disease treatment was noticeably

different, with older patients getting cholecystectomy less often. It was established that the variety of treatment alternatives for older individuals with AC was restricted not by age per se, but rather by disease severity (grade III AC) and/or low physical state (ASA III-IV). Even in elderly individuals with grade I-II AC, laparoscopic cholecystectomy may be done safely and effectively. Patients with grade III AC have a significant risk of morbidity and death, and therapy should be tailored to their specific needs. ASA IV individuals should avoid cholecystectomy, being antimicrobial therapy and cholecystectomy the best choice.

18. Teshaev O.R. et al (2020)[49] compared surgical intervention modalities in patients with acute calculouscholecystitis associated with type 2 diabetes mellitus to establish the best procedure. The surgical solution for acute calculouscholecystitis in individuals with type 2 diabetes mellitus in 195 patients includes LCE for 53.3 percent, oblique muscle-splitting micro approach in the right hypochondrium for 46.7 percent, and open conversion for 38 percent. The use of the laparoscopic approach in the treatment of acute calculouscholecystitis in type 2 diabetes mellitus patients resulted in a statistically significant decrease in operation time and intraoperative blood loss. The comparative examination of the treatment results demonstrated that the use of LCE in diabetic patients offers a number of benefits over the open technique.
19. Hossain M et al (2020)[50] evaluated the clinical characteristics and outcomes of the hospitalised patients with acute pancreatitis (AP) with or without diabetes mellitus (DM) (DM). This research involved 68 participants. They discovered that the key clinical characteristics, laboratory indicators, and serum albumin levels were considerably greater in the diabetic AP group compared to the non-diabetic AP group. DM was related with severe type of AP compared to the non-diabetic group. It has been proposed that diabetes worsens the illness severity of AP.
20. Kaplan U. et al (2021)[51] evaluated the probable difference in the illness process between individuals with diabetes mellitus (DM) and those without. This research comprised a total of 272 patients. they were noticed that the bile cultures were collected from 252 (92.64 percent) patients and were positive in 134 (53.2 percent) patients. Two pathogens were detected in 4% of cases. Blood cultures were taken from 231 individuals and 35 were positive (15.2 percent). The most prevalent isolate was Escherichia coli, which was found in 22.3 percent of positive bile cultures and 40 percent of positive blood cultures. Although diabetic individuals had much more positive bile cultures, the severity of the condition was not greater, according to Tokyo criteria. When diabetes individuals were compared to non-diabetic patients, it was concluded that acute cholecystitis was not more severe nor had a significant difference in bacteriological features.
21. Arora JK et al (2021)[52] examined the impact of co-existing clinical disorders on morbidity and mortality after laparoscopic cholecystectomy. They were noted that the distribution of

complications was similar between patients and controls. (90 percent against 92.50 percent, respectively.) Proportion of patients with complications was substantially lower in individuals without history of prior attack as compared to with history of previous attack. Distribution of complications were similar between diabetes and non-diabetic. it was determined that the presence of co- morbid illnesses in the form of diabetes mellitus and hypertension did not raise the risk of acute post surgical complications. However, a history of past acute cholecystitis might raise the likelihood of intra-operative adhesions around the calots triangle, extending the time of surgery and increasing the risk of post-operative problems.

22. Martínez-Alpuche R.A. et al (2021)[53] examined the postoperative discomfort related to open cholecystectomy in diabetic and non-diabetic individuals. It was shown that diabetes individuals experienced much more intense pain than non-diabetic people. In diabetes and non-diabetic individuals, the mean overall pain score was 7.2 0.3 and 5.3 0.3, respectively. Furthermore, 60 percent of diabetes individuals felt severe pain (NRS > 8) compared to 20 percent of non-diabetics. In both groups, the time to the beginning of postoperative discomfort was around 35 minutes. It was observed that diabetes patients having OC had higher postoperative pain intensity and a higher incidence of patients with severe pain ratings when compared to non-diabetic patients.
23. Serban D. et al. (2021)[54] planned to examine the clinical, paraclinical, intraoperative findings, and postoperative complications in diabetes vs. nondiabetic individuals with acute cholecystitis. They discovered that the diabetic group had higher CCI and ASA risk classifications, with 34.78 percent of patients having three or more related co morbidities. Diabetes had no statistically significant relationships with the severity of the cholecystitis or the chance of conversion. Minor problems, such as surgical site infections, and significant cardiovascular events were more prevalent in the diabetic category, and were well linked with preoperative status and baseline cardiovascular comorbidities. They were concluded that the Laparoscopic cholecystectomy is a safe surgery for diabetic individuals, which may deliver the best results, by lowering the risks of surgical wounds.
24. Lal S et al. (2021)[55] examined the numerous characteristics and results of early and delayed laparoscopic cholecystectomies, as well as their safety and practicality. The conversion rate in this trial was 10% in the early laparoscopic cholecystectomy (ELC) group and 14% in the delayed laparoscopic cholecystectomy (DLC) group. The mean operational time was 77.30± 20.078 minutes vs 66.94± 29.501 minutes in the ELC and DLC groups, respectively; the mean blood loss was 82.60 ±59.67 minutes vs 65.40± 74.21 minutes in the ELC and DLC groups, respectively; Postoperative complication was 4 (8 percent) vs 7 (14 percent) for ELC and DLC

groups, respectively. However, the patients in the ELC group had a considerably shorter hospital stay (4.46 ± 1.32 vs 6.0 ± 2.54 days). They found that in individuals with acute cholecystitis, early cholecystectomy is safe and practical. Early cholecystectomy provides final therapy by avoiding the hazards of failed conservative treatments and recurrent episodes of acute cholecystitis while also shortening the mean hospital stay without increasing morbidity and mortality.

25. Luthra A et al (2022)[56] compared the intraoperative results of diabetics and nondiabetics undergoing elective laparoscopic cholecystectomy. A total of 75 diabetics and 75 nondiabetics were recruited from two groups of individuals with simple gallstone disease. It was discovered that when the groups were compared, the elective laparoscopic cholecystectomy was the most common. There were no demographic differences between the groups. Adverse intraoperative findings such as thick-walled gallbladder (GB), pericholecystic collections, and adhesions to the surrounding structures, surgical problems, modification to subtotal cholecystectomy, and open conversion were more likely in diabetes patients than in nondiabetic patients. They found that diabetes individuals had more unfavourable intraoperative results than nondiabetic patients after laparoscopic cholecystectomy.

MATERIALS AND METHODS

MATERIALS AND METHODS

- Study area: Department of General Surgery, Santosh Medical College &Hospital, Ghaziabad.
- Study design: Prospective observational comparative study
- Study period: Jan 2021 to June2022
- Sample size: 80

Group A: 40

Group B: 40

$$N_{1=} \{z1-^{\alpha/2}*\sqrt{p}*q*(1+1/K)+z1-\beta*\sqrt{p1}*q1+(p2*q2/k)\}^2/\Delta^2$$

P1= (prevalence for diabetic group)
=44.5 P2= (prevalence of non-diabetic
group) = 11.5α = probability of type 1
error = 0.05
power= 0.9 (90%)

Δ= |p2-p1|= absolute difference between two
proportionsn 1= sample size for group1
n 2= sample size for group2

z = critical Z value for a given α or β = 1.96

K = ratio of sample size for group 2 to group 1= 1:1

STUDY TOOLS

CLINICAL FEATURES

1. Right upper quadrant pain similar to biliary colic in onset and character butpersists beyond 4-6hrs.
2. Associated anorexia, nausea and vomiting.
3. Low grade fever.
4. Localised right upper quadrant tenderness.
5. MURPHY'S SIGN positive.
6. Presence of inflammatory mass in right upper quadrant.
7. BOAS'S SIGN POSITIVE - Hyperaesthesia in the right posterior 9 - 11intercostalspace.

INVESTIGATIONS

1. **PBS - Peripheral blood smear**

- To diagnose raised WBC count
- 12000 - 15000 indicate acute cholecystitis
- More than 20,000 indicate complications such as perforation and gangrene.

2. **Biochemical evaluation: -**

- Serum electrolytes - To diagnose co-existent electrolyte abnormalities ifvomiting is present.
- Blood Urea & Creatinine level - To diagnose renal failure if it occurs as a component of multiorgan failure if sepsis ensues and as a routine

preoperativeevaluation.

- Blood sugar level-As a routine investigation and to identify diabetic patients whoare at a high risk of gangrene and perforation of gallbladder.
- HBA1C Glycated hemoglobin-To study the blood sugar control over the lastthree months in diabetic patients.
- Fasting blood sugar
- Post prandial blood sugar
- Liver function tests

3. PLAIN X-RAYABDOMEN

Patients were subjected to routine plain X-Ray Abdomen
to ruleout other causes of acute abdomen like

1. Duodenal ulcer with gas under the diaphragm
2. Calculi in upper renal system
3. Gas in the biliary system,

4. ULTRASOUND OF ABDOMEN

Patients were subjected to ultrasound evaluation on clinical suspicion of acute cholecystitis.The following findings, if present were suggestive of acute cholecystitis

1. Presence of calculi
2. Presence of gall bladder wall edema>3mm
3. Pericholecystic collection
4. Evidence of free fluid in the subhepatic space
5. Presence of calculi obstructing the cystic duct.
6. Presence of adhesions if any
7. Presence of sonographic Murphy's sign

Time of operation: immediate or delayed OT

Type of surgery: Open or Laparoscopic cholecystectomy

Postoperative period: wound healing and complications Eventful or uneventful

Duration of hospital stay: 5-7 days

INCLUSION CRITERIA:

1. Age – upto 70years
2. Newly diagnosed or Known case of diabetes mellitus
3. Any sex

EXCLUSION CRITERIA:

1. Gall bladder cancer
2. Smoking
3. Alcohol
4. Other comorbidities : cardiovascular , hypertension , COPD , bleeding disorders
5. Previous hepatobiliary surgery
6. Immunocompromised patients
7. Patient unfit for surgery
8. Age more than 70
9. Patient not giving consent

RESULTS AND ANALYSIS

RESULTS

Table 1: Age distribution

	Group A	Group B
21-30 years	2	18
	5.0%	45.0%
31-40 years	13	17
	32.5%	42.5%
41-50 years	16	3
	40.0%	7.5%
Above 50 years	9	2
	22.5%	5.0%
	40	40
	100.0%	100.0%
29.598(a) **0.000**		

In this study, patients have been divided into two groups: group A (Diabetic) and group B (non-diabetic). Among diabetic group, majority of the subjects belonged to 41-50 years (40.0%) followed by 31-40 years (32.5%). Among Non-Diabetic group, majority of the subjects belonged to 21-30 years (45.0%) followed by 31-40 years (42.5%). The mean age was significantly more among Diabetic group compared to Non-Diabetic group (p-value <0.05).

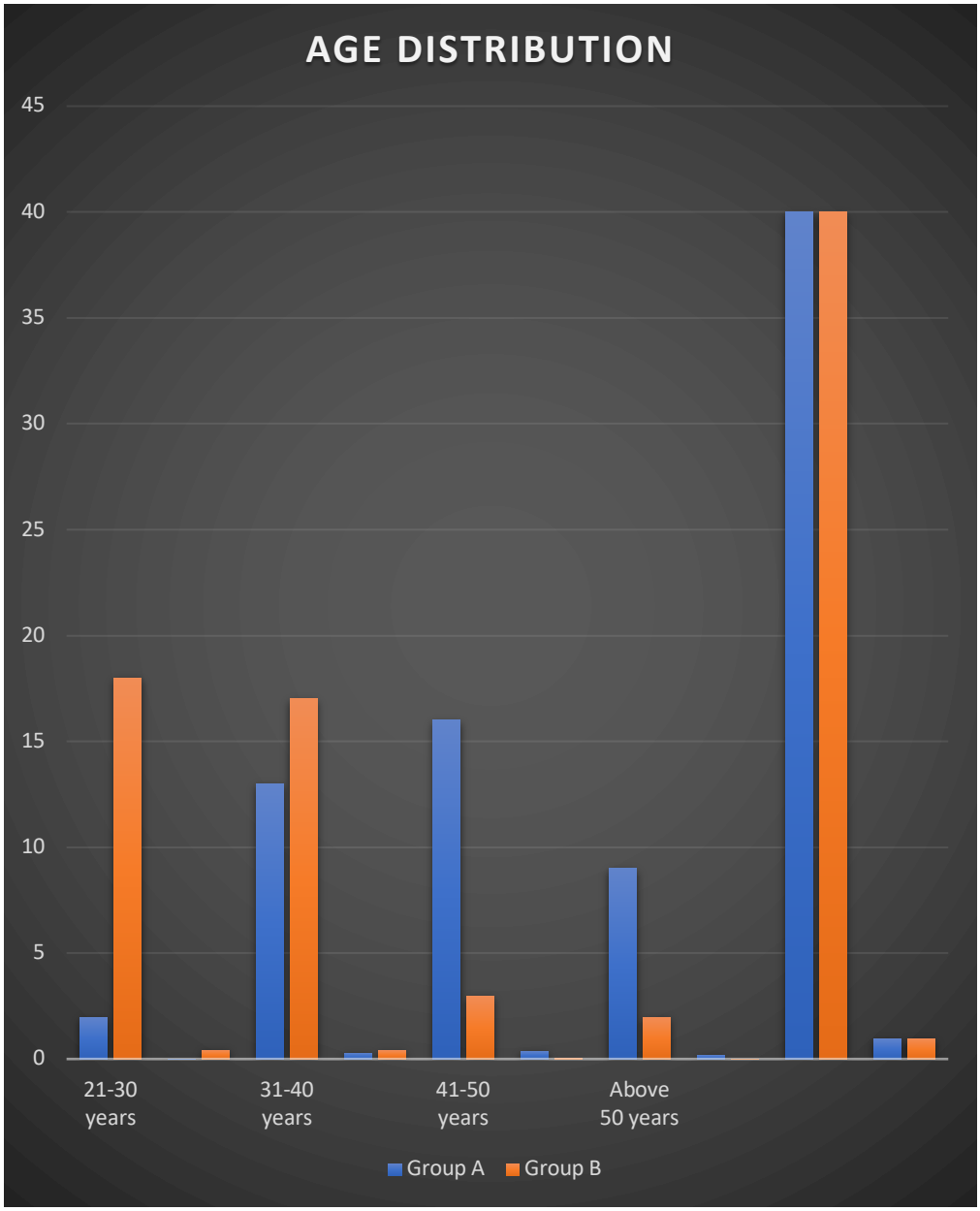


Figure 1: Mean age

Table 2: Gender

	Groups		Total
	Diabetic	Non-Diabetic	
Male	14	9	23
	35.0%	22.5%	28.8%
Female	26	31	57
	65.0%	77.5%	71.3%
p-value = 0.217			

This table is based on the gender distribution of patients : males 14 (35 %), females 26 (65%) in the diabetic group and males 9 (22.5%) and females 31(71.3%) in non-diabetic group. The p value is 0.217 . There was no significant difference in the distribution of males and females between Diabetic and Non-Diabetic groups (p-value >0.05).

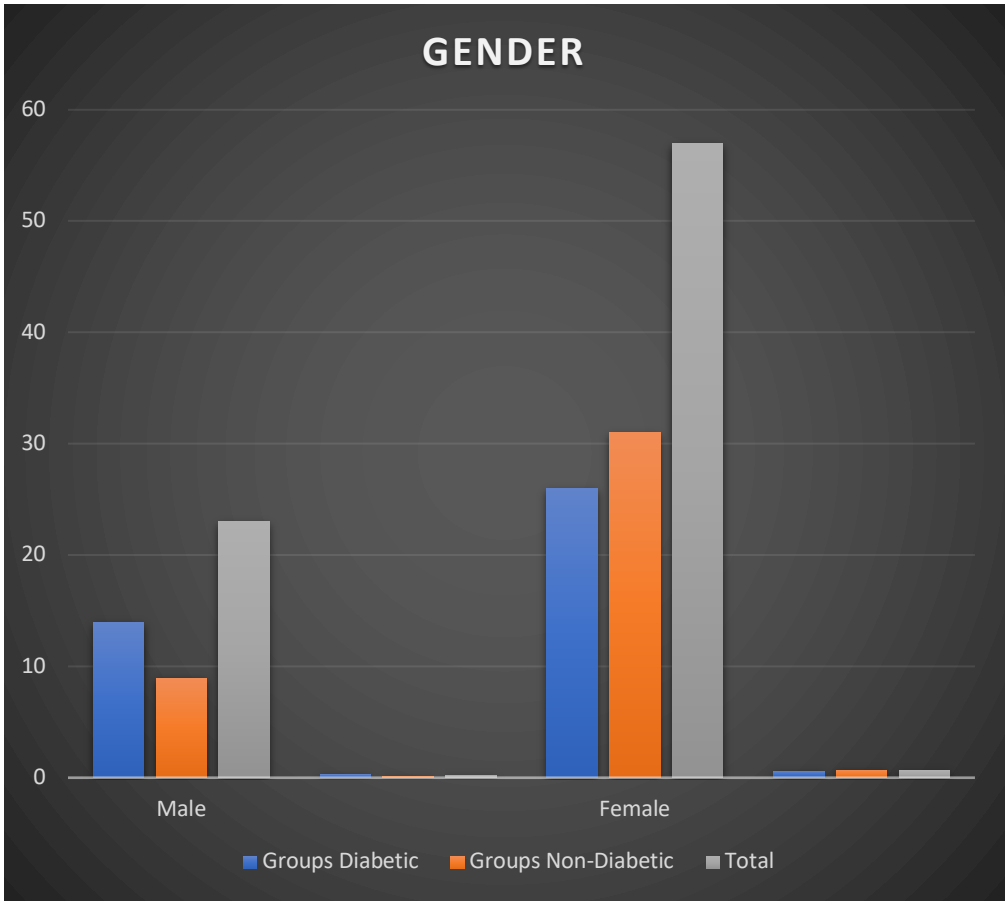


Figure 3: Gender

Table 4: Pulse rate

	Pulse		
Groups	Mean	Std. Deviation	p-value
Non-Diabetic	77.55	7.13	0.172
Diabetic	72.63	21.43	

This table is based on the pulse rate of patients. The non-diabetic group has a mean of 77.55 and a standard deviation of 7.13. The diabetic group has a mean of 72.63 and a standard deviation of 21.43 . The p value is 0.172. There was no significant difference in mean Pulse between Diabetic and Non-Diabetic groups (p-value >0.05)

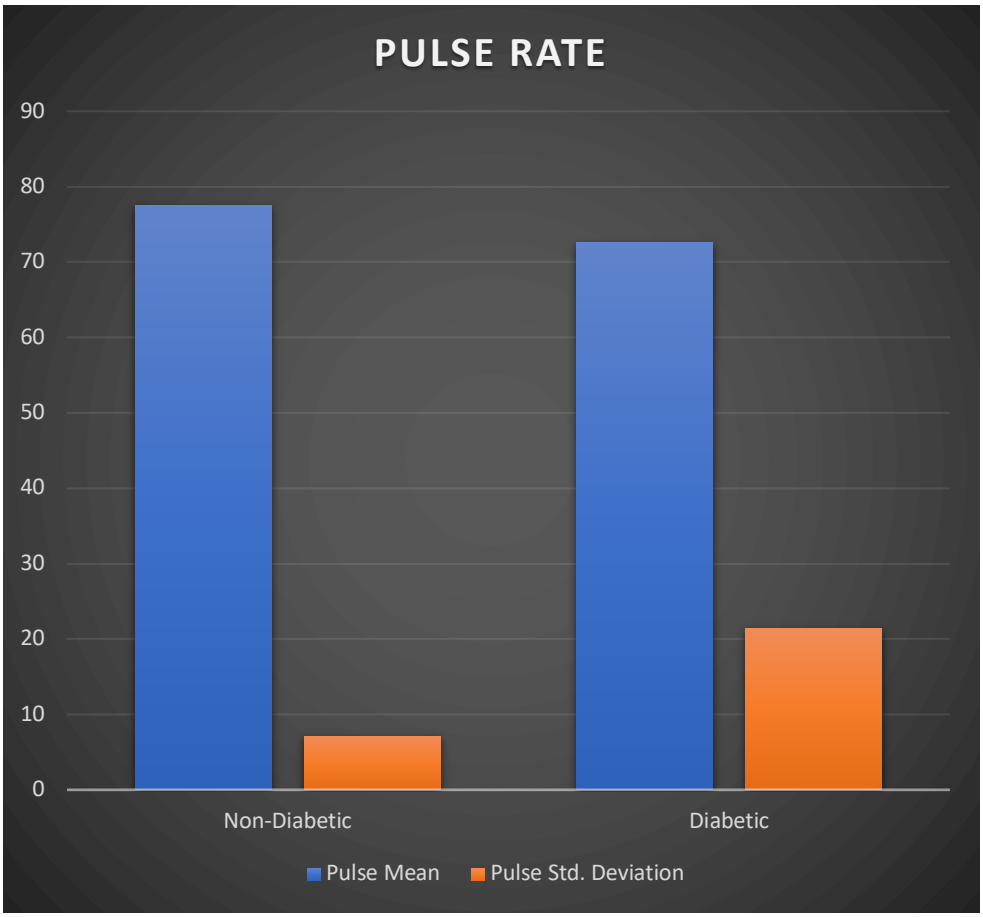


Figure 5: Pulse rate

Table 6: Symptoms

	Groups		p-value
	Diabetic	Non-Diabetic	
Pain	18	10	0.001*
	45.0%	25.0%	
Nausea/ Vomiting	38	35	0.235
	95.0%	87.5%	
Fever	25	16	0.044*
	62.5%	40.0%	

This table is based on symptoms such as pain, nausea / vomiting and fever. In the diabetic group, pain was present in 18 patients and 10 in non-diabetic group with p-value of 0.001. Nausea / vomiting was present in 38 patients in diabetic and 35 in non-diabetic has a p-value of 0.235. Fever was present in 25 patients in diabetic and 16 in non-diabetic patients has a p-value of 0.044. Pain (45 %) and Fever (62.5 %) was significantly more among Diabetic group compared to Non-Diabetic group (p-value <0.05).

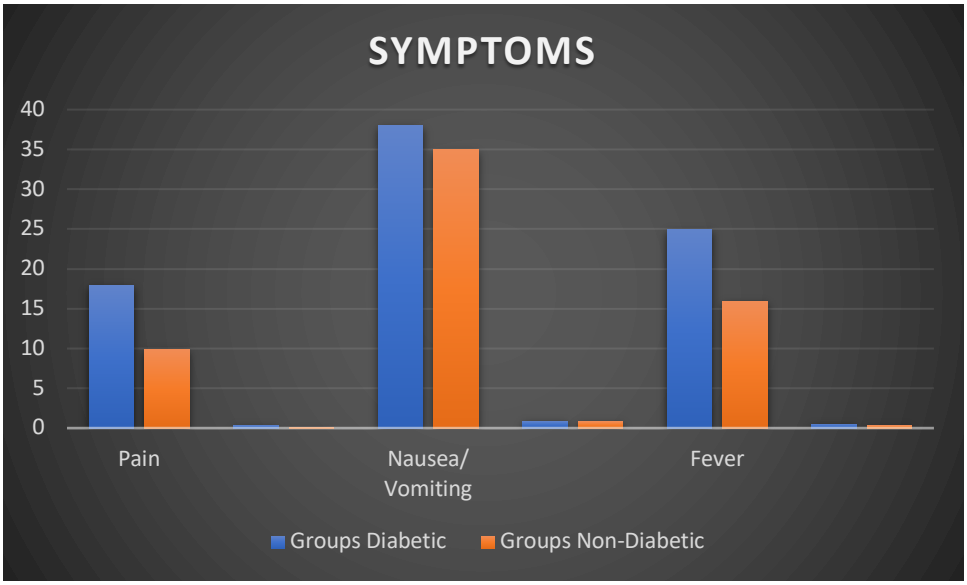


Figure 7: Symptoms

Table 8: Per Abdominal findings

	Groups		p-value
	Diabetic	Non-Diabetic	
Biliary colic	19	20	0.823
	47.5%	50.0%	
Jaundice	3	2	0.021*
	7.5%	5.0%	
Guarding/ Rigidity	7	18	0.008*
	17.5%	45.0%	
Murphy's sign	10	20	0.021*
	25.0%	50.0%	
BOA 'S SIGN	0	0	1.000
	0.0%	0.0%	

This table is based on per abdomen findings such as biliary colic in 3 patients among diabetic group and 2 patients in non-diabetic group with p-value of 0.823, murphy's sign in 20 patients among diabetic group and 8 patients in non-diabetic group with p-value 0.021 and boa's sign 0 out of 80 patients with p-value 1.000. Jaundice (3%) Biliary Colic (47.5%) , guarding and rigidity (17.5%) and Murphy's Sign (25%) was significantly more among Diabetic group compared to Non-Diabetic group. (p-value <0.05)

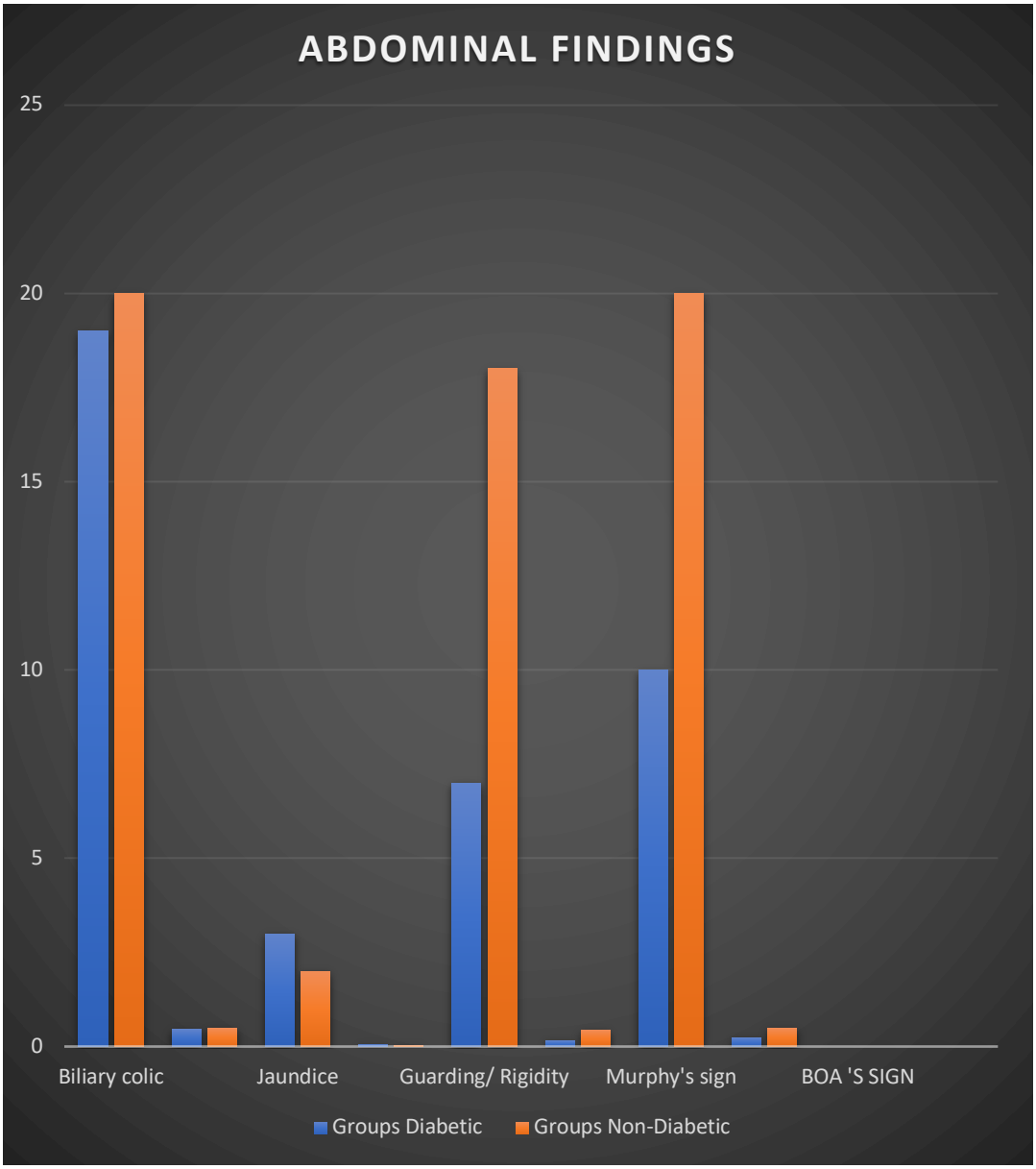


Figure 9: Per Abdominal findings

Table 10: Wound complications

Wound complication	Groups		Total	p-value
	Diabetic	Non-Diabetic		
No	35	39	74	0.041*
	87.5%	97.5%	92.5%	
Yes	5	1	6	
	12.5%	2.5%	7.5%	

This table is based on wound complications where 5 patients in the diabetic group and 1 patient in non diabetic group had complication with p-value 0.041 . Wound complication was significantly more among Diabetic group (12.5%) compared to Non-Diabetic group (2.5%).(p-value <0.05)

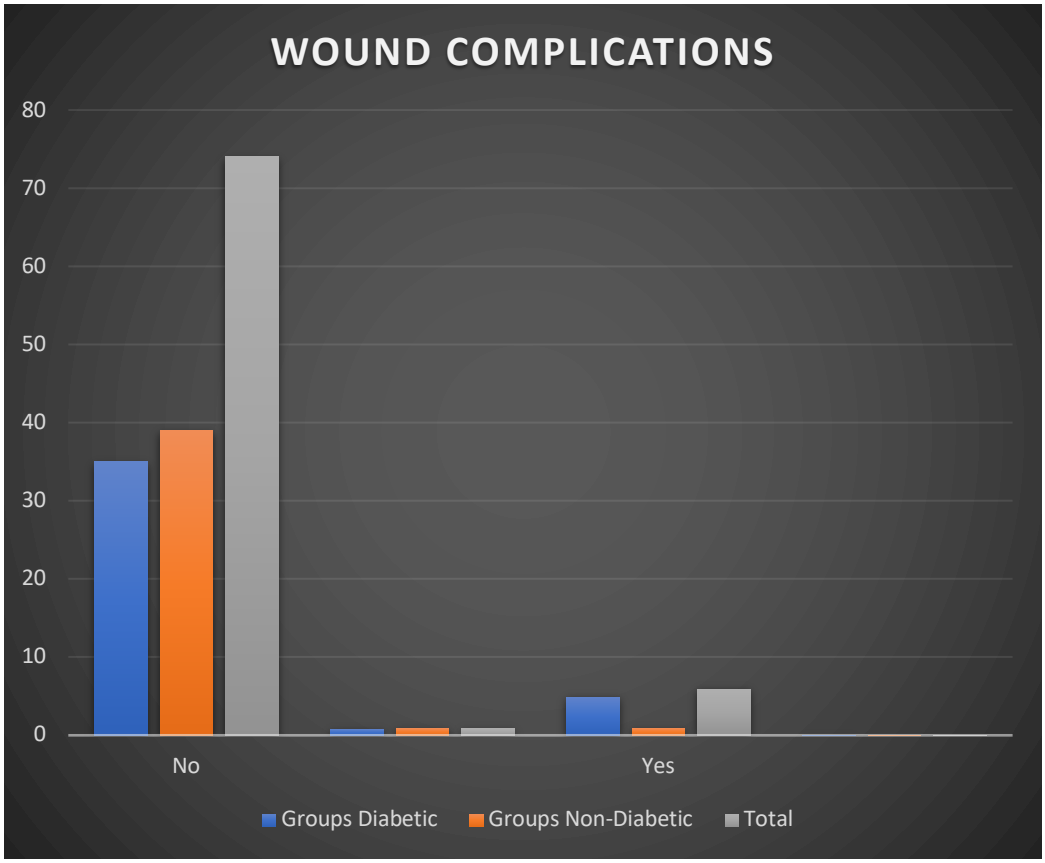


Figure 11: Wound complications

Table 12: Bile Leak

Bile Leak	Groups		Total	p-value
	Diabetic	Non-Diabetic		
No	38	39	77	0.556
	95.0%	97.5%	96.3%	
Yes	2	1	3	
	5.0%	2.5%	3.7%	

This table is based on the bile leak intraoperatively. Bile leak was seen in 2 patients in diabetic group and 1 patient in non-diabetics. Bile Leak did not differ significantly between Diabetic (5%) and Non-Diabetic groups(2.5%). (p-value > 0.05). Bile leak in diabetic patients was because of difficult cholecystectomy, GB fossa leak and slippage of ligature. In non-diabetic it was due to difficult cholecystectomy and GB fossa leak.

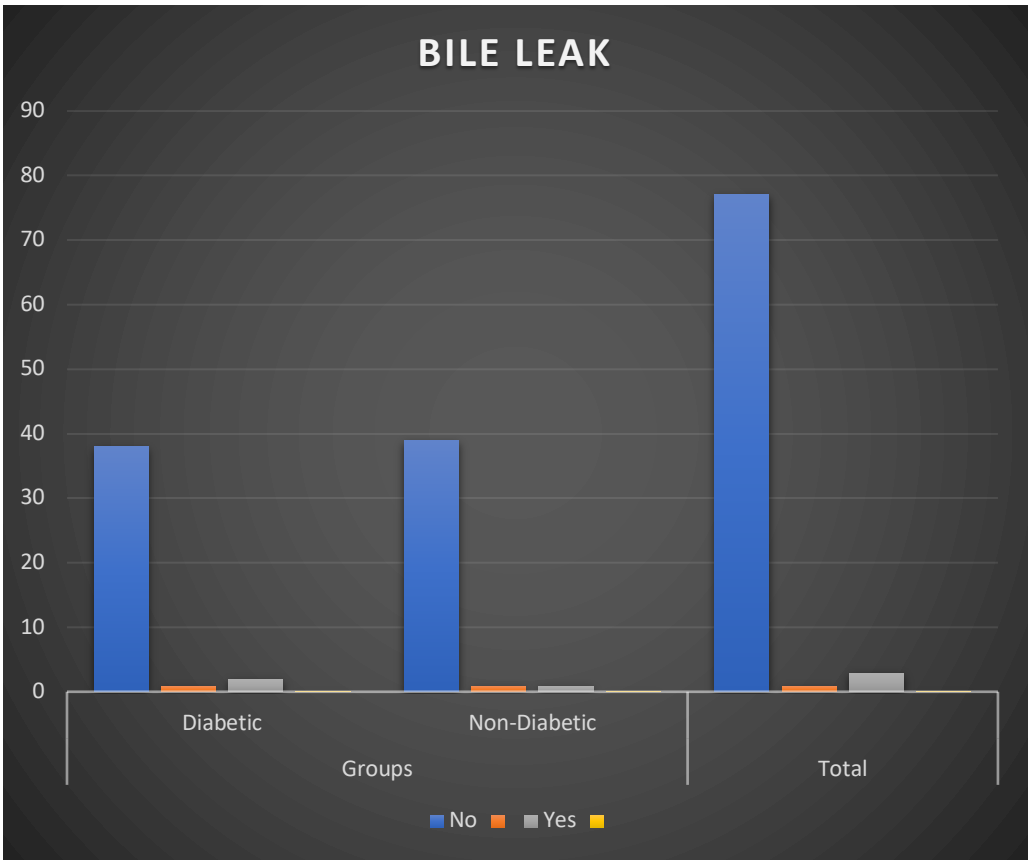


Figure 13: Bile Leak

Table 14: Gall bladder pathology

Gall bladder status	Groups		p-value
	Diabetic	Non-Diabetic	
Normal	8	21	0.046*
	20.0%	52.5%	
Distended	23	16	
	57.5%	40.0%	
Oedematous	5	2	
	12.5%	7.5%	
Mucocele	6	3	
	15.0%	7.5%	
Empyema gall bladder	3	1	
	7.5%	2.5%	
Phyregian cap	1	0	
	2.5%	0.0%	

This is table is based on gall bladder pathology and status such as normal , distended 23 (57.5%)in diabetics and 16 (40%) in non-diabetics with p-value 0.046 . Mucocele is seen in 6 patients in diabetics(15%) and 3 patients in non-diabetics (7.5%) with p-value 0.0001. Empyema gall bladder , oedematous , phyregian cap was found to be more among diabetics as compared to the non-diabetic group . Distended Gall bladder and mucocele was significantly more among Diabetic group. (p-value <0.05)

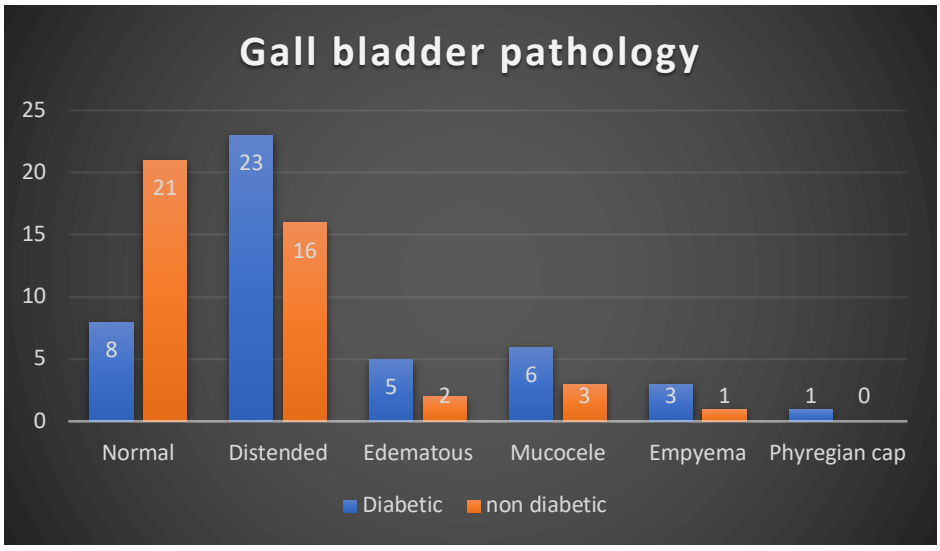


Figure 15: Gall bladder pathology

Table 16: Type of surgery

Lap/ open cholecystectomy	Groups		p-value
	Diabetic	Non-Diabetic	
Lap	33	38	0.035*
	41.3%	95.0%	
Lap to Open	7	2	
	8.8%	5.0%	

This table is based on type of surgery . Lap was done in 33 patients in diabetics and 38 in non diabetics with p-value of 0.035. Lap to open was done in 7 (8.8%) patients in diabetics and 2(5%) in non diabetics. Lap to Open was significantly more among Diabetic group compared to Non-Diabetic group (p-value<0.05)

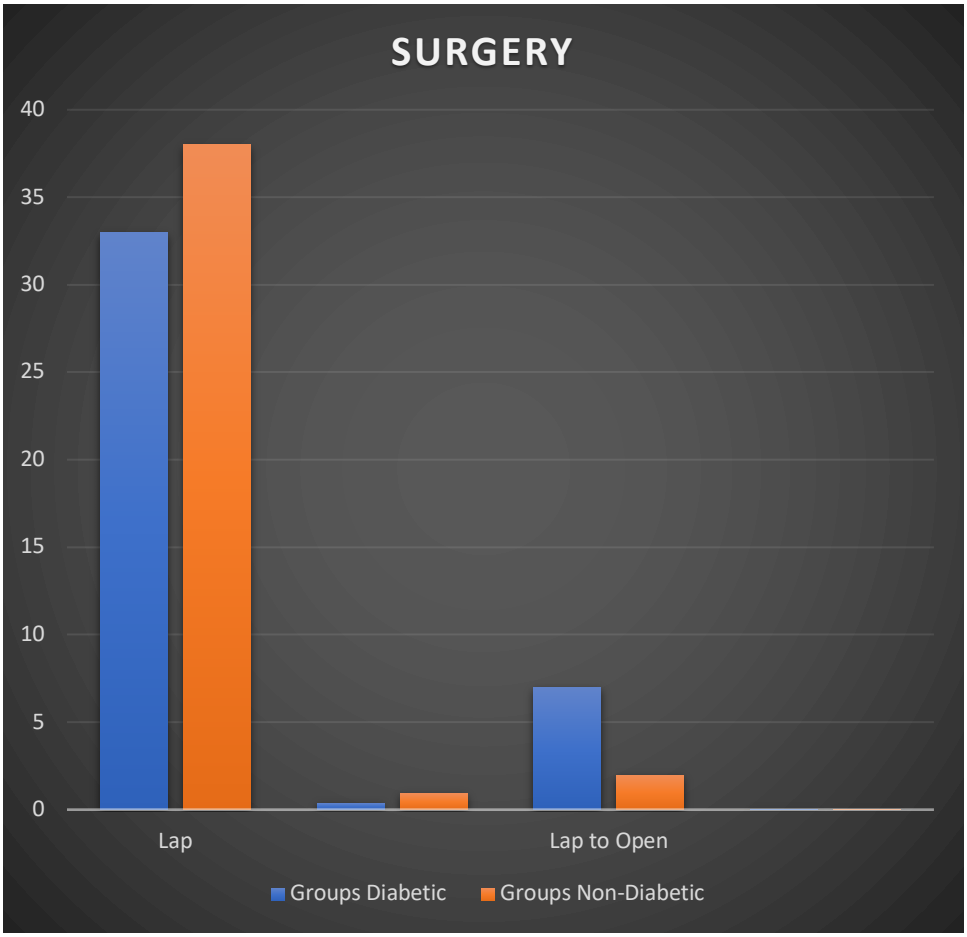


Figure 17: Type of surgery

Table 18: Drain placement

Drain Placed	Groups		p-value
	Diabetic	Non-Diabetic	
No	21	24	0.517
	52.5%	60.0%	
Yes, Subhepatic	19	16	
	47.5%	40.0%	

This table is based on drain placement intra-operatively among the two groups. No drain placement in 21 patients in diabetics and 24 patients in non-diabetics with a p-value of 0.014. Subhepatic drain was placed in 19 patients in diabetics and 16 patients in non-diabetics. Drain Placement was significantly more among Diabetic group compared to Non-Diabetic group.(p-value <0.05)

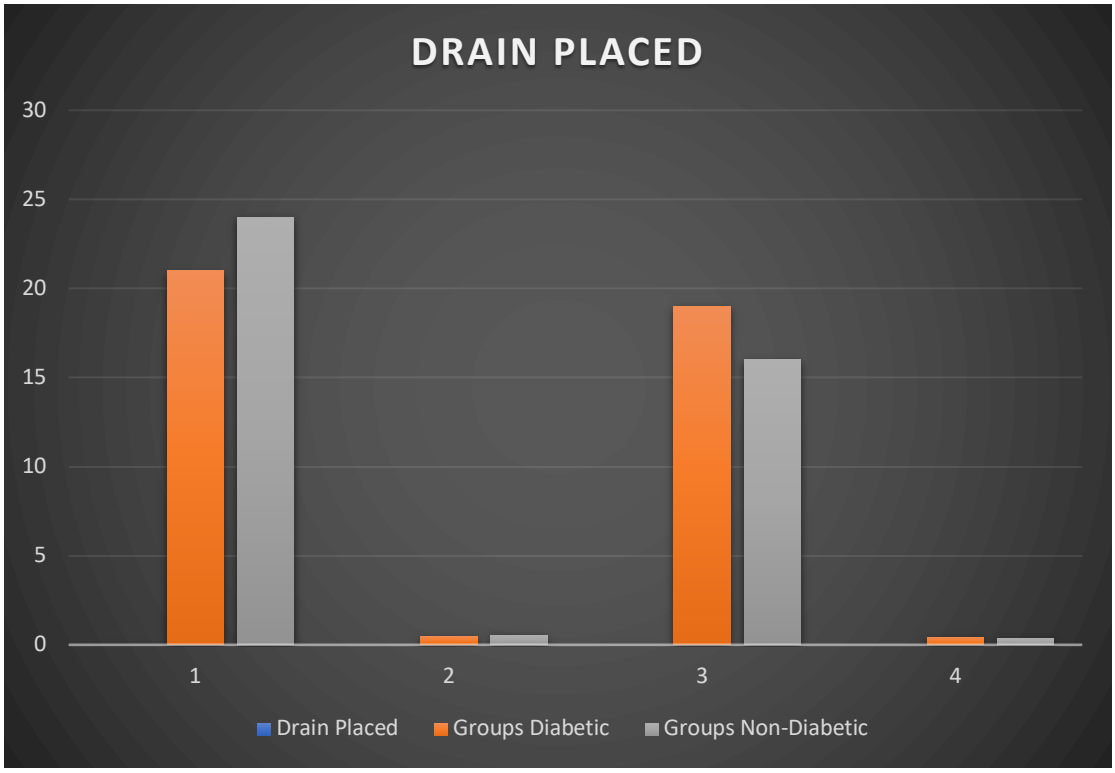


Figure 19: Drain placement

Table 20: Wound Infection

Wound infection	Groups		p-value
	Diabetic	Non-Diabetic	
No	37	38	0.644
	92.5%	95.0%	
Yes	3	2	
	7.5%	5.0%	

This table is based on post-operative wound infection. No wound infection was seen in 37 patients in diabetics and 38 patients in non-diabetics with p-value of 0.644. Wound infection is seen in 3 patients in diabetics group and 2 patients in non- diabetics. Wound infection was not significantly different between Diabetic and Non-Diabetic groups.(p-value <0.05)

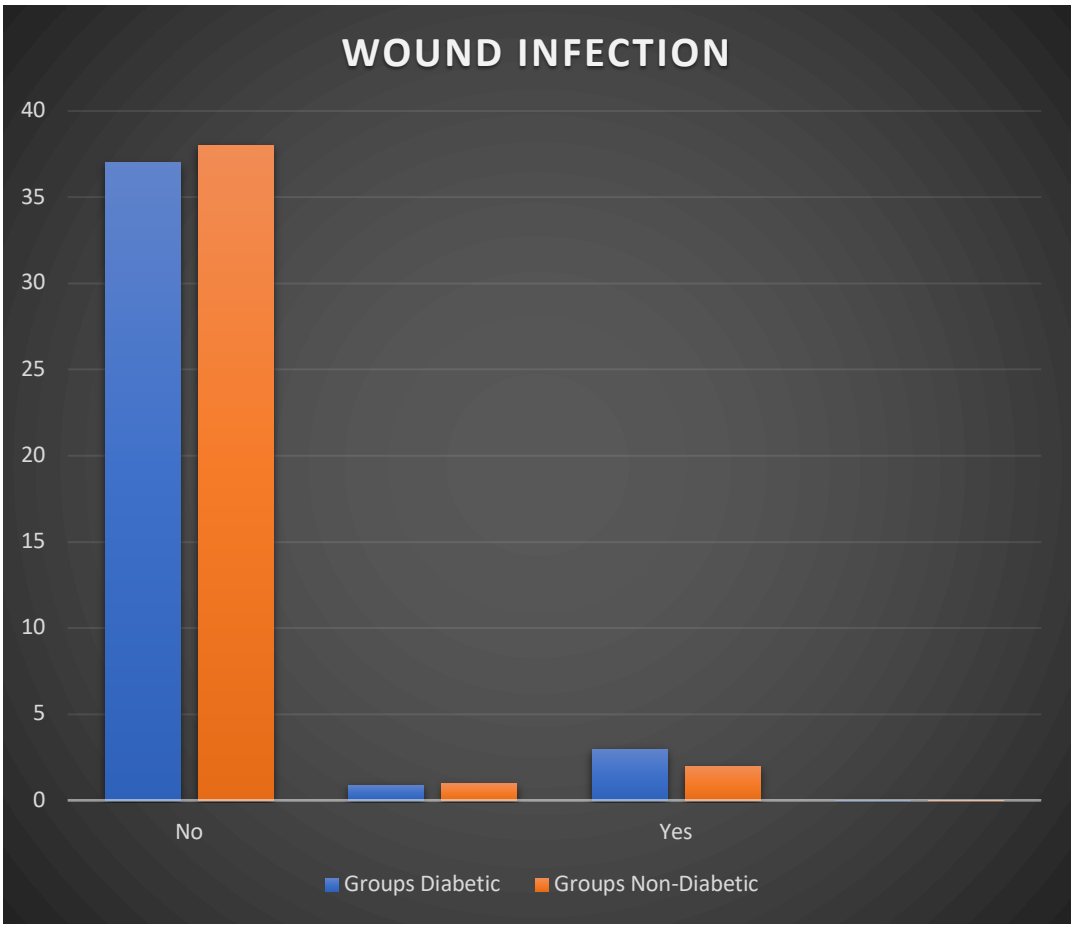


Figure 11: Wound Infection

Table 21: Calot triangle anatomy

Calot's triangle anatomy	Groups		p-value
	Diabetic	Non-Diabetic	
Normal	34	38	0.285
	85.0%	95.0%	
Double cystic duct	1	0	
	2.5%	0.0%	
Frozen, dense pericholecystic adhesions	5	2	
	12.5%	5.0%	

This table is based on calot's triangle anatomy such as normal in 34 patients in diabetic group and 38 patients in non diabetics ; double cystic duct in 1 patients in diabetics and 0 in non-diabetic; frozen ,dense pericholecystic adhesions in 5 patients in diabetics and 2 in non-diabetics with p value of 0.285. No significant difference was between Diabetic and Non-Diabetic groups. (p-value <0.05)

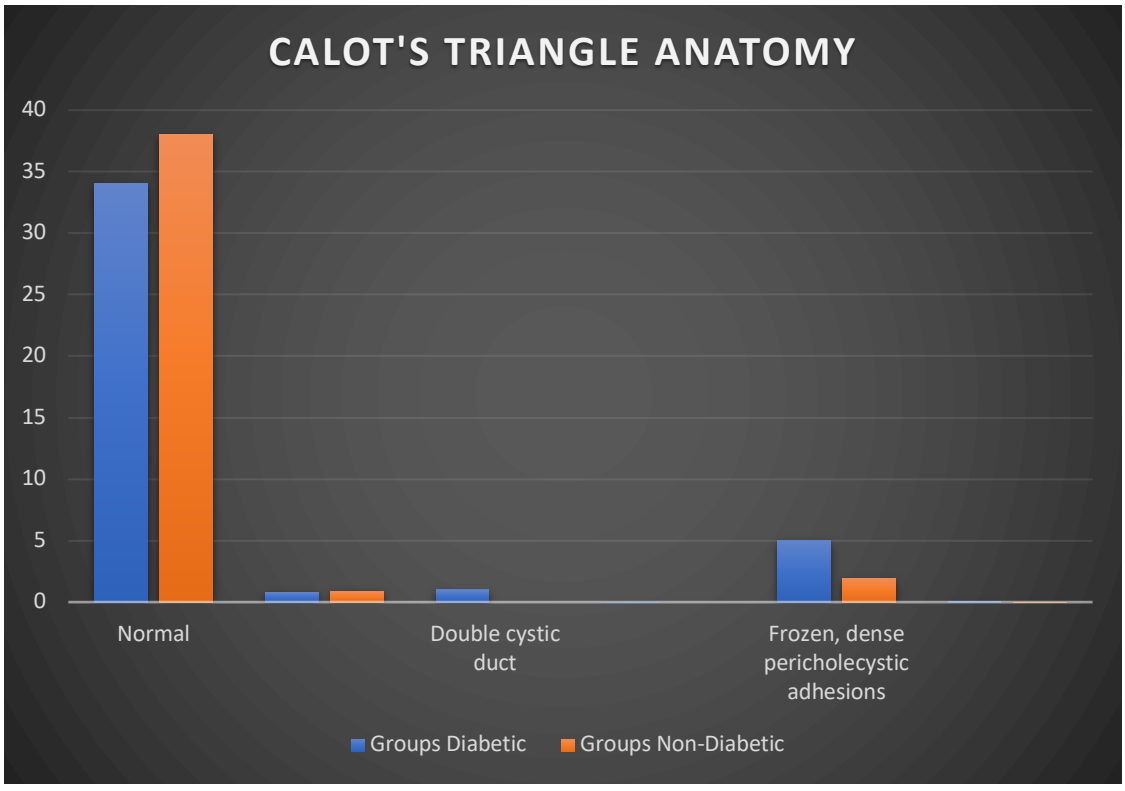


Figure 22: Calot triangle anatomy

Table 23: Number of calculi

Number of Calculus	Groups		p-value
	Diabetic	Non-Diabetic	
One	15	20	0.260
	37.5%	50.0%	
Multiple	25	20	
	62.5%	50.0%	

This table is based on the number of calculi. In diabetic group, 15 patients and 20 patients in non-diabetics has single calculus. 25 patients in diabetics and 20 patients in non-diabetics had multiple calculus. The p-value is 0.260. Multiple Calculus was significantly more among Diabetic group compared to Non-Diabetic group.(p-value <0.05)

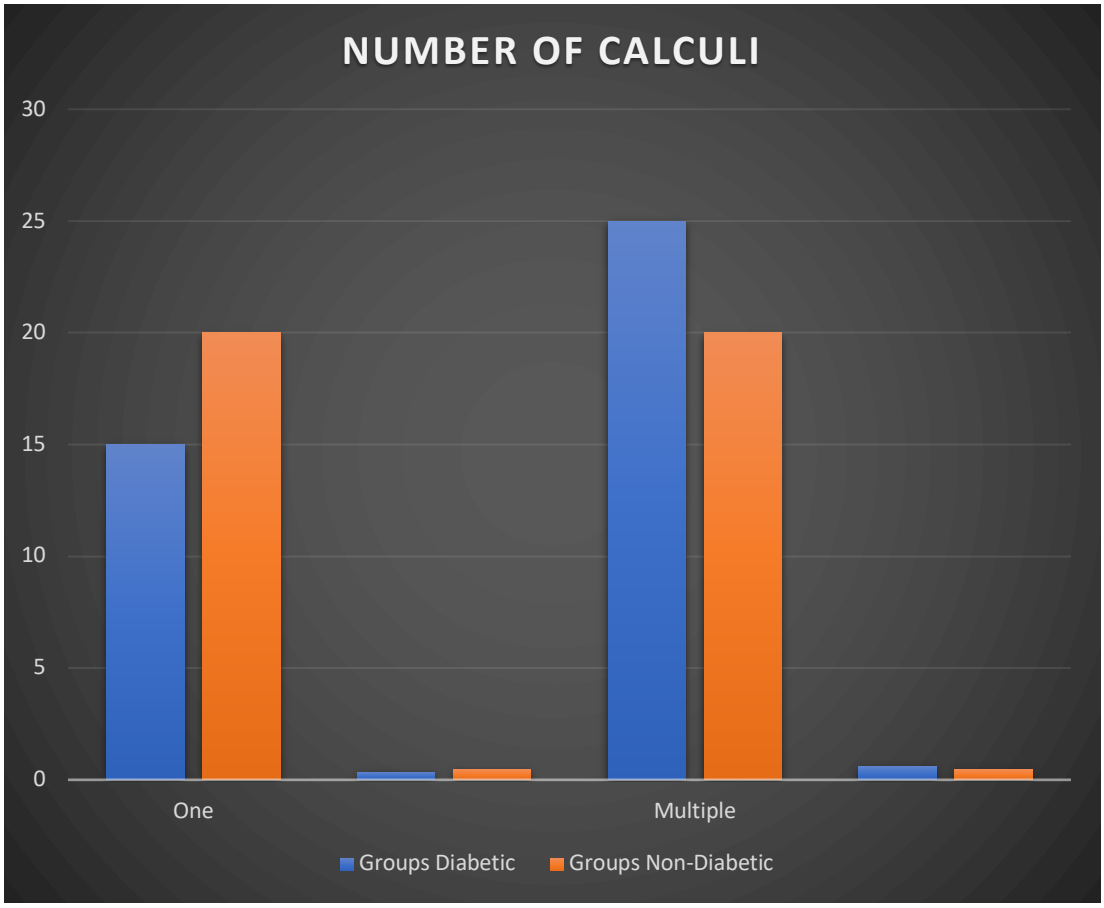


Figure 24: Number of calculi

DISCUSSION AND CONCLUSION

DISCUSSION

Gallstone disease is common in India, affecting more women than men and is increasing with time.[57] This may be related to lifestyle modifications and is more commonly diagnosed due to the ease of availability of ultrasonography to assess abdominal problems.

The most effective method of treating gallstone disease is now laparoscopic cholecystectomy.[58] It is less painful, requires a shorter hospital stay, allows for an earlier return to work, and is more acceptable in terms of appearance. The surgical challenge is increased by the association of advancing age and male sex with more unfavourable intraoperative results.

Age

In present study, the mean age was significantly more among Diabetic group compared to Non-Diabetic group. Age increases the likelihood of having numerous cholecystitis bouts. Vivek et al. experienced difficulty in performing surgery in patients over 65 years due to adhesions.[59]

In our study, patients have been divided into two groups: group A (Diabetic) and group B (non-diabetic). Among diabetic group, majority of the subjects belonged to 41-50 years (40.0%) followed by 31-40 years (32.5%). Among Non-Diabetic group, majority of the subjects belonged to 21-30 years (45.0%) followed by 31-40 years (42.5%). The mean age was significantly more among Diabetic group compared to Non-Diabetic group.

Gender

It was found in our research that there was no significant difference in the distribution of males and females between Diabetic and Non-Diabetic groups. *Kaplan et al*.[51] has shown similar findings in his study . Male sex was a statistically significant predictor of inflammation and surgery was more challenging in a gender-specific research by Ambe et al.[60]

The number of patients who were hospitalized for Acute cholecystitis in the general population, among them 28.8% of patients those who underwent Cholecystectomy were men. Male gender has been identified as a risk factor for severe cholecystitis by *Lein et al*.[61] This data indicates increased disease severity in males and may support the suggestion of early intervention in male patients with acute cholecystitis . The reason for doing Cholecystectomy would be severity or lack of improvement in symptoms[60]. In our study , majority of the patients were female.

In our study based on the gender distribution of patients : there were 14 males , 26 were females in the diabetic group and 9 were males and 31 were females in non-diabetic group. There was no

significant difference in the distribution of males and females between Diabetic and Non-Diabetic groups.

Pain score

In our study, pain was significantly more among Diabetic group compared to Non-Diabetic group. *Martínez-Alpuche R.A. et al*.[53] reported that diabetic individuals experienced much more intense pain than non-diabetic people. In diabetic and non-diabetic individuals, the mean pain score was 7.2 and 5.3 respectively. Furthermore, 45 percent of diabetic individuals felt severe pain (NRS > 8) compared to 25 percent of non-diabetics. It was observed that diabetic patients having open cholecystectomy had higher postoperative pain intensity when compared to non-diabetic patients.

Our findings are consistent with those of *Karci and co-workers*,[62] who found that diabetic patients having major elective abdominal surgeries experienced greater pain and needed more rescue analgesics than non-diabetic patients. Similar to this, diabetic patients with knee osteoarthritis have more pain than non-diabetics.[63] Additionally, diabetic patients' pain thresholds were lower than those of non-diabetic patients in the oral mucosa, at the tips of the fingers, and in the toes.[64]

Additionally, diabetic patients with glycosylated haemoglobin (HbA1c) levels higher than 6.5% are more likely to experience inadequate analgesia in the early postoperative period[65] or require more analgesics during surgery,[66] which suggests that these patients experience more pain than non-diabetic patients with HbA1c levels below 6.5%. When considered together, it may be predicted that pain relief for diabetic patients may decline with uncontrolled diabetes indicated by high HbA1c levels.[67]

Open cholecystectomy patients who are non-diabetics are reported to experience moderate to severe postoperative discomfort.[68,69] Polanco-Garca et al., demonstrated that the mean pain score following general surgery was about 6 NRS (severe pain), and the percentage of patients who experienced moderate to severe pain was approximately 40%.[70] Although both diabetic and non-diabetic individuals experienced moderate to severe postoperative pain, diabetic patients were more likely to experience severe postoperative pain. According to the findings of the previous clinical investigations,[63] diabetes exacerbates the pain hypersensitivity associated with surgery, and this is also consistent with the findings of several preclinical studies.[71-73]

Postoperative discomfort is linked to tissue damage brought on by the operation. Persistent pain and hyperalgesia are frequently brought on by tissue injury. There is growing evidence to support the idea that both peripheral and central sensitization cause chronic pain and hyperalgesia. According to experimental models of tissue damage, diabetic animals exhibit more hyperalgesia than non-diabetic animals.[71,72,74,75] .

Symptoms

Symptoms such as pain, nausea / vomiting and fever have been described in this study. In the diabetic group, pain was present in 18 patients and 10 in non-diabetic group with p-value of 0.001. Nausea / vomiting was present in 38 patients in diabetic and 35 in non-diabetic has a p-value of 0.235. Fever was present in 25 patients in diabetic and 16 in non-diabetic patients has a p-value of 0.044. Pain and Fever were significantly more among Diabetic group compared to Non-Diabetic group (p-value <0.05) .

Signs

Jaundice, Biliary Colic and Murphy's Sign was significantly more among Diabetic group compared to Non-Diabetic group. This was similar to the findings by Serban et al., Lal et al and Luthra et al.[54-56] . In our study , on per abdomen examination , findings such as biliary colic was found in 3 patients among diabetic group and 2 patients in non-diabetic group with p-value of 0.823, murphy's sign was found in 20 patients among diabetic group and 8 patients in non-diabetic group with p-value 0.021. Jaundice, Biliary Colic and Murphy's Sign was significantly more among Diabetic group compared to Non-Diabetic group. (p-value <0.05)

Wound complications

We found that wound complication was significantly more among Diabetic group compared to Non-Diabetic group. *Serban D. et al*.[54] stated that minor problems, such as surgical site infections, and significant cardiovascular events were more prevalent in the diabetic category, and were well linked with preoperative status and baseline cardiovascular comorbidities.

In our study , wound complications where found in 5 patients in the diabetic group and 1 patient in non-diabetic group had complication with p-value 0.041 . Wound complication was significantly more among Diabetic group compared to Non-Diabetic group.(p-value <0.05)

Bile Leak

Bile Leak was significantly more among Diabetic group compared to Non-Diabetic group. In this study bile leak was seen in 2 patients in diabetic group and 1 patient in non-diabetics group intraoperatively . Bile leak in diabetic patients was because of difficult cholecystectomy, GB fossa leak and slippage of ligature. In non-diabetic it was due to difficult cholecystectomy and GB fossa leak.

Gall bladder pathology

Distended Gall bladder was significantly more among Diabetic group. *Serban D. et al*.[54] discovered that the diabetic group had higher Charlson co-morbidity index (CCI) and ASA risk classifications.

In our study the gall bladder pathology and status have been studied . It was found that the gall bladder was distended in 39 patients in the diabetics and 29 patients in non-diabetics group with p-value of 0.046 . Mucocele was seen in 6 patients in diabetics and 3 patients in non-diabetics with p-value 0.0001. Empyema gall bladder , oedematous gall bladder, phyregian cap was found to be more among diabetics as compared to the non-diabetic group . Distended Gall bladder and mucocele was significantly more among Diabetic group. (p-value <0.05)

Type of surgery : Lap or Lap to Open

Lap to Open conversion of surgery was significantly more among Diabetic group compared to Non-Diabetic group. *Luthra A et al*.[56] discovered that open conversion were more likely in diabetic patients than in non-diabetic patients. *Serban D. et al*.[54] discovered that the rate of conversion was higher in the diabetic group (13.2%) vs. the non-diabetic group (6.9%)

The majority of the existing research points at diabetes mellitus as a risk factor for switching from laparoscopic to open cholecystectomy. Diabetic patients with higher HbA1c levels had a considerably greater rate of conversion. The degree of biliary disease and conversion risk are impacted by the patient's ability to manage their diabetes. Patients with diabetes appear to experience postoperative complications more frequently.[46]

In our study the type of surgery performed was laparoscopic cholecystectomy or conversion of lap to open cholecystectomy . Lap was done in 33 patients in diabetics and 38 in non-diabetics group with p-value of 0.035. Lap to open was done in 7 patients in diabetics and 2 in non-diabetics. Lap to Open was significantly more among Diabetic group compared to Non-Diabetic group (p-value<0.05)

Drain Placement

Drain Placement was significantly more among Diabetic group compared to Non-Diabetic group. *Kaplan U et al*.[51] noticed that the bile cultures were collected from 252 (92.64 percent) patients and were positive in 134 (53.2 percent) patients. Two pathogens were detected in 4% of cases. Blood cultures were taken from 231 individuals and 35 were positive (15.2 percent). The most prevalent isolate was Escherichia coli, which was found in 22.3 percent of positive bile cultures and 40 percent of positive blood cultures. *Hossain M et al*.[50] stated that laboratory indicators, and serum albumin levels were considerably greater in the diabetic group compared to the non-diabetic group.

In our study the drain placement intra-operatively was seen among the two groups. No drain placement in 21 patients in diabetics and 24 patients in non-diabetics with a p-value of 0.014. Subhepatic drain was placed in 19 patients in diabetics and 16 patients in non-diabetics group . Drain Placement was significantly more among Diabetic group compared to Non-Diabetic group.(p-value <0.05).

The drain placement was done sub-hepatically due to oozing from the gall bladder fossa or due to any suspicion of leak or following any bile leak or after abdominal wash during surgery.

Wound infection

In this current study, wound infection was significantly more among the Diabetic group as compared to Non-Diabetic group. *Serban D. et al*.[54] discovered that minor problems, such as surgical site infections, and significant cardiovascular events were more prevalent in the diabetic category, and were well linked with preoperative status and baseline cardiovascular comorbidities.

In our study post-operative wound infection was evaluated . No wound infection was seen in 37 patients in diabetics and 38 patients in non-diabetics with p-value of 0.644. Wound infection is seen in 3 patients in diabetics group and 2 patients in non- diabetics. Wound infection was not significantly different between Diabetic and Non-Diabetic groups.(p-value <0.05)

Perioperative complications

In this current study, frozen, dense pericholecystic adhesions was significantly more among Diabetic group compared to Non-Diabetic group. Multiple Calculus was significantly more among Diabetic group compared to Non-Diabetic group. In our study calot's triangle anatomy was normal in 34 patients in diabetic group and 38 patients in non-diabetics ; double cystic duct was seen in 1 patients in diabetics and 0 in non-diabetic; frozen ,dense pericholecystic adhesions was seen in 5 patients in diabetics and 2 in non-diabetics with p value of 0.285. No significant difference was between Diabetic and Non-Diabetic groups. (p-value <0.05).

In our study based on the number of calculi , in diabetic group, 15 patients and 20 patients in non-diabetics had single calculus. 25 patients in diabetics and 20 patients in non-diabetics group had multiple calculus. Multiple Calculus was significantly more among diabetic group compared to non-diabetic group.(p-value <0.05).

Ismat U et al.[44] found in his study that 3 patients in group A developed a surgical site infection. In group B, 2 patients had surgical site infection, and it was established that the presence of diabetes mellitus did substantially impact the development of surgical site infection in individuals having laparoscopic cholecystectomy.

Luthra A et al.[56] discovered that adverse intraoperative findings such as thick-walled gallbladder (GB), pericholecystic collections, and adhesions to the surrounding structures, surgical complications and modification to subtotal cholecystectomy were more likely in diabetic patients than in non-diabetic patients.

Poorly managed diabetes (elevated HbA1c > 6) was associated with a higher probability of switching to an open procedure, according to a research by Ibrahim et al. in Singapore.[76] They believed that improperly managed blood sugar levels cause significant inflammation and, in turn, severe adhesions, which affect patients' anatomy of the hepatobiliary structures. Additionally, it was shown that individuals who underwent conversion had much higher postoperative complication rates. *Walsh et al*.[16] showed no appreciable increase in surgical morbidity in diabetics. They believed that the main reason diabetics had higher morbidity was because they are older and have other medical issues.

Arora JK et al.[52] noted that the distribution of complications was similar between patients and controls. (90 percent against 92.50 percent, respectively.) Proportion of patients with complications was substantially lower in individuals without history of prior attack as compared to those with history of previous attack. Distribution of complications were not similar between diabetes and non-diabetic. It was found that a history of past attack of acute cholecystitis might raise the likelihood of intra-operative adhesions around the calots triangle, extending the time of surgery and increasing the risk of post-operative problems.

Through a variety of mechanisms, including the glycosylation of complement proteins, inhibition of immunoglobulin-mediated opsonization of bacteria, inhibition of neutrophil migration, phagocytosis, and apoptosis, chronically elevated blood sugar levels alter the immune response and make the diabetic patients more susceptible to infections. Therefore, maintaining strict glycemic control between 100 and 140 mg/ml is essential for reducing healthcare-associated infections and promoting rapid healing of surgical sites.[54]

SUMMARY AND CONCLUSION

The study findings can be summarized as follows:

- The mean age was significantly more among Diabetic group compared to Non-Diabetic group. Among diabetic group, majority of the subjects belonged to 41-50 years (40.0%) followed by 31-40 years (32.5%). Among Non-Diabetic group, majority of the subjects belonged to 21-30 years (45.0%) followed by 31-40 years (42.5%)
- There was no significant difference in the distribution of males and females between Diabetic and Non-Diabetic groups.
- There was no significant difference in mean Pulse between Diabetic and Non-Diabetic groups.
- Pain was significantly more among Diabetic group compared to Non-Diabetic group. Pain (45 %) and Fever (62.5 %) was significantly more among Diabetic group compared to Non-Diabetic group (p-value <0.05).
- Jaundice (3%) Biliary Colic (47.5%) , guarding and rigidity (17.5%) and Murphy's Sign (25%) was significantly more among Diabetic group compared to Non-Diabetic group. (p-value <0.05)
- Wound complication was significantly more among Diabetic group (12.5%) compared to Non-Diabetic group(2.5%). (p-value <0.05)
- Bile Leak did not differ significantly between Diabetic (5%) and Non-Diabetic groups(2.5%). (p-value > 0.05).
- Gall bladder was found to be distended in (57.5%)in diabetics and (40%) in non-diabetics with p-value 0.046 . Mucocele is seen in diabetics(15%) and in non-diabetics (7.5%) with p-value 0.0001. Distension and mucocele was significantly found to be more in diabetics group as compared to non-diabetic group. (p-value <0.05)
- Lap to Open was significantly more among Diabetic group (8.8 %) compared to Non-Diabetic group(5%).(p-value <0.05)
- Drain Placement was significantly more among Diabetic group (47.5%)compared to Non-Diabetic group(40 %).
- Wound infection was not significantly more among Diabetic group (7.5%)compared to Non-Diabetic group(5%). (p-value >0.05)

- Frozen, dense pericholecystic adhesions was significantly more among Diabetic group (12.5%) compared to Non-Diabetic group (5%).
- Multiple Calculus was significantly more among Diabetic group (62.5%) compared to Non-Diabetic group (50%).

Conclusion

Compared to non-diabetics, elective laparoscopic cholecystectomy in diabetics was associated with more unfavourable intraoperative findings, including thicker gallbladder, pericholecystic adhesions, and constricted gallbladder. Additionally, it was shown that the diabetic group required greater time for anaesthetic recovery and conversion to open. In contrast to the age- and sex-matched control groups discovered in this investigation, elective surgery does not seem to have a poor prognosis in people with well-controlled diabetes. Men experience greater difficulties than women when having the same operation.

To have a successful result, rigorous preoperative planning and meticulous intraoperative surgical methods using expertise and judgment are essential. The drawback of the study was that enough patients were not involved. As a result, further research using a larger sample size is necessary to support a conclusive finding.

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PROFORMA

Name:

Age:

Sex:

Address:

Income:

IPD No.:

Occupation:

Presenting
Complaints: Right
upper quadrant pain
Biliary colic

Nausea /
Vomiting
Fever, with
chills
Jaundice

Biliary Colic

Previous Surgeries

Hypertension / diabetes Miletus / asthma / thyroid disorders / Tb / COPD.
Oral contraceptive usage

Smoking / Alcohol

Past History

Personal History
ON EXAMINATION

Vital Signs - PR, BP, Temp, RR

Other Systems

CVS

CNS

RS

Per abdomen

Guarding/rigidity

Right hypochondrium tenderness

Murphy's sign

Boas' sign

Right hypochondriac mass abdomen

INTRA OP. FINDINGS: (ELECTIVE /EMERGENCY /EARLY)

Cholecystectomy / Cholecystectomy / Lap. CholecystostomyGall bladder status

Calot's triangle anatomy CBD size, stones Others Number and type of stones Whether drain placed

POST OP. COURSE:

Drain removal

Suture Removal

Wound infection

Wound

Complication

Respiratory complications

Bile leak

GB Histology

SANTOSH MEDICAL COLLEGE AND HOSPITAL (GHAZIABAD)

INFORMED CONSENT

I_____Son/Daughter/Wife of _____ residentof hereby declare that I give my full, free and voluntary informed consent in the thesis "**Comparative study of outcomes of acute cholecystitis among diabetic and non-diabetic patients**"**.** Dr._____has informed me to myfull satisfaction, in the language I understand, about the purpose, nature of study and various laboratory investigations for the study to be carried out. I have been informed about the duration of the study and instructions to be followed during the study period.

I give full consent for being enrolled in the above study and I reserve my rights to withdrawfrom this study, whenever I wish without prejudice of my right to undergo further treatmentat Santosh Hospital, Ghaziabad U.P.

Patient's/ Patient's relative's signature

Doctorssignature or thumb impression

Name: Date:

We have witnessed that the patient signed the above form in the presence of his free willafter fully understanding its content.

Signature of
WitnessName

Signature of
InvestigatorName

Dr. Priyanka Jaiswal

Designation: PG
Department ofGeneral
Surgery
Contact No.-8897755948

संतोष मेडिकल कॉलेज एवं अस्पताल (गाजियाबाद)

<u>सहमतिपत्र (सूचित)</u>

मैं _____ बेटा / बेटी /पत्नी_____

निवासी _____ घोषणा करता हूं कि मैं

डॉ ._____, ने मुझे मेरी भाषा में अपने अध्ययन **Comparative study of outcomes of acute cholecystitis among diabetic and non- diabetic patients** अध्ययन के उद्देश्य के बारे में ,और अध्ययन के दौरान अपनाई जानेवाली विभिन्न प्रयोग शाला जांच के बारे में सूचित कर दिया है ।मुझे अध्ययन की अवधि और उसके दौरान अपनाए जाने वाले निर्देश भी मेरी भाषा मे समझा दिए गए हैं |मैं उपरोक्त अध्ययन में बिना किसी दबाव के दाखिला ले रहाहूं और पूर्ण सहमति देरहाहूं | सन्तोष अस्पताल, गाजियाबाद में अपने आगे के उचार पर बिना प्रति कूल प्रभाव डाले इस अध्ययन से और प्रयोग शाला जांच की प्रकृति से मैं जब चाहूँ बाहर जाने के अपने अधिकार को सुरक्षित रख रहा हूं |

रोगी / रोगी के रिश्तेदार के हस्ताक्षर　　डॉक्टर के हस्ताक्षर

नाम

दिनांक:

मेरी उपस्थिति में मरी जने पूरी तरह से डाकटर के बताएगए अध्ययन और निर्देशों को समझने के बाद अपनी स्वतंत्र इच्छा से ऊपर के फार्म पर हस्ताक्षर किए हैं।

हस्ताक्षर　　नाम: डॉप्रियंका जायसवाल

पदनाम: पीजी

शल्य चिकित्सा विभाग

संपर्कनंबर-8897755948

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INTRODUCTION

BIOSTATISTICS CERTIFICATE

TO WHOM SO EVER IT MAY CONCERN

CERTIFICATE

This is to certify that the results of the research work titled **"A COMPARATIVE STUDY OF OUTCOMES OF ACUTE CHOLECYSTITIS AMONG DIABETIC AND NONDIABETIC PATIENTS"** carried out by **Dr. Priyanka Jaiswal** from Department of Surgery, Santosh Medical College & Hospital, Ghaziabad, U.P. was analyzed statistically using software SPSS version 25.0 (IBM SPSS Statistics, IBM corp., 2018).

The necessary statistical tests were applied as per the requirement of the thesis and to the best of my knowledge.

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ETHICAL CLEARANCE

SANTOSH
Deemed to be University
(Established u/s 3 of the UGC Act, 1956)

F. No. SU/2021/1830[44] Dated: 17.07.2021

Subject: Grant of Ethical Clearance for Thesis Protocol Topic **"OUTCOMES OF ACUTE CHOLECYSTITIS IN DIABETIC AND NON DIABETIC PATIENTS",** submitted by **Dr. Priyanka Jaiswal,** MD/MS student of Batch 2020-2021, Department of **General Surgery**.

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With reference to his/her request for grant of Ethical Clearance for the Thesis Protocol entitled **"OUTCOMES OF ACUTE CHOLECYSTITIS IN DIABETIC AND NON DIABETIC PATIENTS", Dr. Priyanka Jaiswal** is informed that the Thesis Protocol submitted by him/her was considered by the **Screening Committee** of the Santosh Medical College & Hospital in its meetings held on 19.03.2021, 22.03.2021, 24.03.2021, 25.03.2021, 26.03.2021, 27.03.2021 and 06.04.2021. The recommendations of the **Screening Committee** were considered in detail by the Institutional **Ethics Committee** in its meeting held on **12.07.2021** and the same was **approved** by the **Ethics Committee**.

He /She is informed accordingly for further necessary action.

[Dr. V. P. Gupta]
REGISTRAR

SANTOSH DEEMED TO BE UNIVERSITY ★ REGISTRAR ★ GHAZIABAD, NCR DELHI

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Copy to:

1. The Vice Chancellor
2. The Dean, SMC&H
3. The Dean – Research
4. The Dean – Academics
5. The Director - IQAC
6. Dr. Rajeev Verma (Guide)
7. HOD of **General Surgery**

MASTERCHART

NAME	AGE	DIAGNOSIS	SEX	PARI	NAUSEA/VOMITIN G	FEVER	DURATION OF SYMPTOMS	VIRAL MARKERS	COMORBID CONDITION	GUARDING/RIGIDITY	PRESENTING QUADRNT PULSE	JAUNDICE	BILIARY COLIC	MURPHY'S SIGN	BOA 'S SIGN	WOUND COMPLICATION	BILE LEAK	GALL BLADDER STATUS	DURATION OF SURGERY	LAP/OPEN CHOLECYSTECTOMY	DRAIN PLACED OR NOT	WOUND INFECTION	CALOT'S TRIANGLE ANATOMY	NUMBER OF CALCULUS	OOZING/BLEEDING FROM GB FOSSA
MRS SUDHESH	45	ADHOMYOMYOMATOSIS	F	0	1	0	3 Days	NEGATIVE	DIABETES- 5 YEARS	1	PHC	82	1 YES	1 NO	1 NO		1 YES	CHOLEMATOUS	INCREASED	LAP TO OPEN	YES, SUBHEPATIC	YES		DOUBLE CYSTIC DUCT	1	
AJAY PATHAK	38	CHOLELITHIASIS	F	0	1	1	2 day	NEGATIVE	DIABETES- 5 YEARS	1	PHC	78	1 YES	1 NO	1 NO		1 YES	CHOLEMATOUS	INCREASED	LAP TO OPEN	YES, SUBHEPATIC	YES		NORMAL		multiple		
MRS SHANKUNTALA	45	CHOLELITHIASIS	F	0	1	1	3 Days	NEGATIVE	DIABETES- 3 YEARS	1	PHC	67	1 YES	1 NO	1 NO		1 NO	CHOLEMATOUS	INCREASED	LAP TO OPEN	YES, SUBHEPATIC	YES		PERICHOLECYSTIC		multiple		
MR CHANDRAHAN	35	ACUTE CALCULOUS CHOLECYSTITIS	M	0	1	1	4 Days	NEGATIVE	DIABETIC -20YRS	1	PHC	90	1 YES	1 NO	1 NO		1 NO	EMPHYSEMATOUS GALL BLADDER	INCREASED	LAP TO OPEN	YES, SUBHEPATIC	NONE		PERICHOLECYSTIC		multiple		
MRS MEENAKSHI	32	ACUTE CALCULOUS CHOLECYSTITIS	F	0	1	1	2 Days	NEGATIVE	DIABETES- 2 YEARS	1	PHC	70	1 YES	1 NO	1 NO		1 NO	EMPHYSEMATOUS GALL BLADDER	INCREASED	LAP TO OPEN	YES, SUBHEPATIC	NONE		PERICHOLECYSTIC			1	
MRS VIMMI	30	CHOLELITHIASIS	F	0	1	1	3 Days	NEGATIVE	DIABETES- 2 YEARS	1	PHC	72	1 YES	1 NO	1 NO		1 NO	EMPHYSEMATOUS GALL BLADDER	INCREASED	LAP TO OPEN	YES, SUBHEPATIC	NONE		PERICHOLECYSTIC		multiple		
MR MANVEER	45	CHRONIC CHOLECYSTITIS WITH CHOLELITHIASIS	M	0	1	0	5 days	NEGATIVE	DIABETES- 3 YEARS	0	PHC	80	0 YES	1 NO	0 NO		0 NO	DISTENDED, MUCOCELE, THICKENED GB WALL	INCREASED	LAP TO OPEN	YES, SUBHEPATIC	NONE		PERICHOLECYSTIC ADHESIONS		multiple		1
MRS POOJA	50	CHOLELITHIASIS	F	1	1	0	2 days	NEGATIVE	HYPERTENSION -3YRS	1	PHC	84	0 YES	1 NO	0 NO		0 NO	CHOLEMATOUS	NORMAL	LAP	YES, SUBHEPATIC	NONE		PERICHOLECYSTIC		multiple		1
MRS RANJANA	42	CHOLELITHIASIS	F	1	1	0	3 Days	NEGATIVE	NONE	1	PHC	68	1 YES	1 NO	0 NO		0 NO	EMPHYSEMATOUS GALL BLADDER	INCREASED	LAP	YES, SUBHEPATIC	NONE		PERICHOLECYSTIC		multiple		1
MR AMIT	33	CHOLELITHIASIS	M	1	0	0	1 day	NEGATIVE	NONE	1	PHC	90	1 YES	1 NO	1 NO		1 YES	CHOLEMATOUS	NORMAL	LAP	YES, SUBHEPATIC	NONE		NORMAL		multiple		1
MRS SAVINA	55	WITH CHOLELITHIASIS WITH DIAPEMA	F	0	1	0	2 day	NEGATIVE	DIABETES- 3 YEARS	0	PHC	89	0 YES	1 NO	0 NO		1 NO	NORMAL	NORMAL	LAP	YES, SUBHEPATIC	NONE		NORMAL			1	
MRS SUDHA	31	CHOLELITHIASIS	F	1	0	0	3 Days	NEGATIVE	NONE	0	PHC	88	0 YES	1 NO	0 NO		0 NO	DISTENDED	NORMAL	LAP	YES, SUBHEPATIC	NONE		NORMAL		multiple		1
MR SANJAY	32	WITH MUCOCELE	M	1	1	0	5 days	NEGATIVE	NONE	1	PHC	78	0 YES	1 NO	0 NO		0 NO	MUCOCELE	INCREASED	LAP TO OPEN	YES, SUBHEPATIC	NONE		NORMAL			1	
MRS JYOTI	26	CHOLELITHIASIS	F	1	1	0	5 days	NEGATIVE	NONE	1	PHC	76	0 YES	1 NO	0 NO		0 NO	DISTENDED	NORMAL	LAP	YES, SUBHEPATIC	YES		NORMAL		multiple		1
MRS SUNITA	32	CHOLELITHIASIS	F	1	0	0	2 day	NEGATIVE	NONE	1	PHC	74	0 NO	1 NO	0 NO		0 NO	CHOLEMATOUS	NORMAL	LAP	YES, SUBHEPATIC	NONE		NORMAL		multiple		1
MR GOMATH PRASAD	65	ACUTE CALCULOUS CHOLECYSTITIS	M	0	1	0	1 month	NEGATIVE	DIABETIC -20YRS	0	PHC	75	0 YES	1 NO	0 NO		0 NO	CHOLEMATOUS	NORMAL	LAP	YES, SUBHEPATIC	NONE		NORMAL		multiple		1
MRS NIRMALA DEVI	55	CHOLELITHIASIS	F	1	1	0	2 days	NEGATIVE	NONE	1	PHC	70	0 NO	1 NO	0 NO		0 NO	CHOLEMATOUS	NORMAL	LAP	YES, SUBHEPATIC	NONE		NORMAL		multiple		1
MRS ALKA VERMA	30	CHOLELITHIASIS	F	1	0	0	1 day	NEGATIVE	NONE	1	PHC	65	0 YES	1 NO	0 NO		0 NO	DISTENDED	NORMAL	LAP	YES, SUBHEPATIC	NONE		NORMAL		multiple		1
MRS ANAMIKA	28	CHOLELITHIASIS	F	1	1	0	5 days	NEGATIVE	NONE	1	PHC	80	0 YES	1 NO	0 NO		0 NO	DISTENDED	NORMAL	LAP	YES, SUBHEPATIC	NONE		NORMAL			1	1
MRS SONI	30	CHOLELITHIASIS	F	1	1	0	6 days	NEGATIVE	NONE	1	PHC	90	0 NO	1 NO	0 NO		0 NO	DISTENDED	NORMAL	LAP	YES, SUBHEPATIC	NONE		NORMAL			1	1
MR SANDEEP	32	CHOLELITHIASIS	M	0	1	0	3 months	NEGATIVE	DIABETES- 3 YEARS	0	PHC	78	0 NO	1 NO	0 NO		0 NO	DISTENDED, PYOCELE	INCREASED	LAP	YES, SUBHEPATIC	NONE		NORMAL		multiple		1
Ms Jayaanti	54	CHOLECYSTITIS WITH	F	1	1	1	3 Days	NEGATIVE	NONE	1	PHC	72	0 NO	1 NO	0 NO		0 NO	DISTENDED	NORMAL	LAP	YES, SUBHEPATIC	NONE		NORMAL			1	1
Mrs Sunita	40	CHOLECYSTITIS	F	0	1	1	2 months	NEGATIVE	DIABETES- 4 YEARS	1	PHC	74	0 NO	1 NO	0 NO		0 NO	CHOLEMATOUS	NORMAL	LAP	YES, SUBHEPATIC	NONE		NORMAL			1	1
Mrs Jyoti	32	CHOLECYSTITIS	F	1	1	1	1 MONTH	NEGATIVE	NONE	1	PHC	76	0 NO	1 NO	0 NO		0 NO	CHOLEMATOUS	NORMAL	LAP	YES, SUBHEPATIC	NONE		NORMAL		Multiple		1
Mr Ahash	23	CHOLECYSTITIS	M	1	1	1	3 DAYS	NEGATIVE	NONE	1	PHC	68	0 NO	1 NO	0 NO		0 NO	CHOLEMATOUS	INCREASED	LAP	YES, SUBHEPATIC	NONE		NORMAL			1	1
Mrs Daya	55	CHOLELITHIASIS	M	0	1	1	5 DAYS	NEGATIVE	DIABETES- 3 YEARS	1	PHC	78	0 NO	1 NO	0 NO		0 NO	CHOLEMATOUS	NORMAL	LAP	YES, SUBHEPATIC	NONE		NORMAL		Multiple		1
Ms Sunita	45	CHOLELITHIASIS	F	0	1	1	20 DAYS	NEGATIVE	DIABETES- 3 YEARS	1	PHC	74	0 NO	1 NO	0 NO		0 NO	DISTENDED	NORMAL	LAP	pES, SUBHEPATIC	NONE		NORMAL		Multiple		1
Mrs Shalini	30	CHOLELITHIASIS	F	0	1	1	1 YEAR	NEGATIVE	NONE	1	PHC	73	0 NO	1 NO	0 NO		0 NO	DISTENDED	NORMAL	LAP	NO	NONE		NORMAL			1	0
Mr Nitin	23	CHOLECYSTITIS	M	0	1	1	3 DAYS	NEGATIVE	NONE	1	PHC	60	0 NO	1 NO	0 NO		0 NO	DISTENDED	NORMAL	LAP	NO	NONE		NORMAL			1	0
Mr Kishnini Devi	57	CHOLELITHIASIS	F	1	1	1	6 MONTHS	NEGATIVE	DIABETES- 2 YEARS	0	PHC	70	0 NO	1 NO	0 NO		0 NO	DISTENDED	NORMAL	LAP	YES, SUBHEPATIC	NONE		NORMAL		Multiple		0
Pinky Mahlaga	36	CHOLECYSTITIS	F	1	1	1	2 day	NEGATIVE	NONE	1	PHC	80	0 NO	1 NO	0 NO		0 NO	DISTENDED	NORMAL	LAP	NO	NONE		NORMAL		Multiple		0
Pooja Raghav	25	CHOLECYSTITIS	F	1	1	1	1 day	NEGATIVE	NONE	1	PHC	82	0 NO	1 NO	0 NO		0 NO	DISTENDED	NORMAL	LAP	NO	NONE		NORMAL			1	0
Mrs Vimala	32	CHOLECYSTITIS	F	0	1	1	2 DAYS	NEGATIVE	DIABETES- 5 YEARS	1	PHC	84	0 NO	1 NO	0 NO		0 NO	DISTENDED	NORMAL	LAP	YES, SUBHEPATIC	NONE		NORMAL		Multiple		1
Mrs Sudha	39	CHOLECYSTITIS	F	0	1	0	5 days	NEGATIVE	DIABETES- 2 YEARS	0	PHC	86	0 NO	1 NO	0 NO		0 NO	DISTENDED	NORMAL	LAP	YES, SUBHEPATIC	NONE		NORMAL		Multiple		1
Mr Mahalakta	24	CHOLECYSTITIS	F	1	1	1	2 day	NEGATIVE	NONE	0	PHC	86	0 NO	1 NO	0 NO		0 NO	DISTENDED	NORMAL	LAP	NO	NONE		NORMAL			1	0
Mrs Reema	29	CHOLECYSTITIS	F	1	1	1	2 DAYS	NEGATIVE	NONE	0	PHC	76	0 NO	1 NO	0 NO		0 NO	DISTENDED	NORMAL	LAP	YES, SUBHEPATIC	NONE		NORMAL		Multiple		1
Mr Balveer singh	59	CHOLECYSTITIS	M	0	1	1	3 Days	NEGATIVE	DIABETES- 5 YEARS	1	PHC	70	0 NO	1 NO	0 NO		0 NO	DISTENDED	NORMAL	LAP	YES, SUBHEPATIC	NONE		NORMAL		Multiple		1
Mr Shubham	29	CHOLECYSTITIS	M	1	1	1	2 day	NEGATIVE	NONE	0	PHC	80	0 YES	1 NO	0 NO		0 NO	DISTENDED	NORMAL	LAP	NO	NONE		NORMAL			1	0
Mr Rajesh	37	CHOLELITHIASIS	F	1	1	1	6 MONTHS	NEGATIVE	NONE	1	PHC	80	0 YES	1 NO	0 NO		0 NO	DISTENDED	NORMAL	LAP	NO	NONE		NORMAL			1	0
Mr Anandha	45	CHOLECYSTITIS WITH	M	0	1	1	20 DAYS	NEGATIVE	DIABETES- 2 YEARS	0	PHC	80	0 YES	1 NO	0 NO		0 NO	DISTENDED	NORMAL	LAP	YES, SUBHEPATIC	NONE		NORMAL		Multiple		1
Meenaji Sharma	30	CHOLECYSTITIS	M	1	1	1	4 Days	NEGATIVE	NONE	0	PHC	79	0 YES	1 NO	0 NO		0 NO	DISTENDED	NORMAL	LAP	NO	NONE		NORMAL			1	0
Poonam Devi	40	CHOLECYSTITIS	F	1	1	1	5 days	NEGATIVE	NONE	0	PHC	86	0 YES	1 NO	0 NO		0 NO	DISTENDED	NORMAL	LAP	NO	NONE		NORMAL			1	0
Mrs Asha	50	CHOLECYSTITIS	F	1	1	1	15 DAYS	NEGATIVE	DIABETES- 3 YEARS	0	PHC	77	0 YES	1 NO	0 NO		0 NO	DISTENDED	NORMAL	LAP	NO	NONE		NORMAL		Multiple		0
Rajesh	54	CHOLECYSTITIS	M	0	1	1	2 day	NEGATIVE	DIABETES- 3 YEARS	0	PHC	79	0 YES	1 NO	0 NO		0 NO	NORMAL	NORMAL	LAP	NO	NONE		NORMAL		Multiple		0
Mr Rahi	34	CHOLECYSTITIS	F	0	1	1	6 DAYS	NEGATIVE	DIABETES- 5 YEARS	0	PHC	72	0 YES	1 NO	0 NO		0 NO	NORMAL	NORMAL	LAP	NO	NONE		NORMAL			1	0
Mr Anil Kumar	29	CHOLECYSTITIS	M	1	1	1	3 Days	NEGATIVE	NONE	0	PHC	75	0 YES	1 NO	0 NO		0 NO	DISTENDED	NORMAL	LAP	NO	NONE		NORMAL			1	0
Raju Kumar	43	CHOLECYSTITIS	M	1	1	1	7 DAYS	NEGATIVE	NONE	0	PHC	76	0 YES	1 NO	0 NO		0 NO	DISTENDED	NORMAL	LAP	NO	NONE		NORMAL			1	0
Bhawna Rawat	32	CHOLECYSTITIS	F	1	1	1	6 days	NEGATIVE	NONE	0	PHC	77	0 YES	1 NO	0 NO		0 NO	DISTENDED	NORMAL	LAP	NO	NONE		NORMAL		Multiple		0
Raj Kumari	35	CHOLECYSTITIS	F	1	1	1	1 DAY	NEGATIVE	NONE	0	PHC	80	0 YES	1 NO	0 NO		0 NO	NORMAL	NORMAL	LAP	NO	NONE		NORMAL			1	0
Mrs Madhu	26	CHOLECYSTITIS	F	1	1	1	1 day	NEGATIVE	NONE	0	PHC	82	0 NO	1 NO	0 NO		0 NO	DISTENDED	NORMAL	LAP	NO	NONE		NORMAL		Multiple		0
Kauser	27	CHOLECYSTITIS	F	1	1	1	3 DAYS	NEGATIVE	NONE	0	PHC	84	0 NO	1 NO	0 NO		0 NO	DISTENDED	INCREASED	LAP TO OPEN	YES, SUBHEPATIC	NONE		NORMAL		Multiple		1
Dimple	28	CHOLECYSTITIS	F	0	1	1	2 DAYS	NEGATIVE	DIABETES- 3 YEARS	0	PHC	88	0 NO	1 NO	0 NO		0 NO	DISTENDED	NORMAL	LAP	YES, SUBHEPATIC	NONE		NORMAL		Multiple		1
Mrs Kaushal	45	CHOLECYSTITIS	F	0	1	1	7 DAYS	NEGATIVE	DIABETES- 5 YEARS	0	PHC	86	0 NO	1 NO	0 NO		0 NO	DISTENDED	NORMAL	LAP	NO	NONE		NORMAL			1	0
Pushpa	34	CHOLECYSTITIS	F	1	1	1	1 DAY	NEGATIVE	NONE	0	PHC	80	0 NO	1 NO	0 NO		0 NO	DISTENDED	NORMAL	LAP	NO	NONE		NORMAL			1	0
Shivnarshi	40	CHOLELITHIASIS	F	0	1	1	6 MONTHS	NEGATIVE	DIABETES- 3 YEARS	0	PHC	70	0 NO	1 NO	0 NO		0 NO	DISTENDED	NORMAL	LAP	NO	NONE		NORMAL		Multiple		0
Gayatri Rani	55	CHOLECYSTITIS	M	0	1	1	10 DAYS	NEGATIVE	DIABETES- 2 YEARS	0	PHC	78	0 NO	1 NO	0 NO		0 NO	DISTENDED	NORMAL	LAP	NO	NONE		NORMAL			1	0
Surender Kumar	42	CHOLECYSTITIS	M	0	1	1	5 DAYS	NEGATIVE	DIABETES- 2 YEARS	0	PHC	79	0 NO	1 NO	0 NO		0 NO	DISTENDED	NORMAL	LAP	NO	NONE		NORMAL		Multiple		0
Jyoti Sharma	38	CHOLECYSTITIS	F	1	1	1	3 DAYS	NEGATIVE	NONE	0	PHC	76	0 NO	1 NO	0 NO		0 NO	DISTENDED	NORMAL	LAP	NO	NONE		NORMAL		Multiple		0
Mamti Devi	46	CHOLECYSTITIS	F	1	1	1	6 DAYS	NEGATIVE	DIABETES- 3 YEARS	0	PHC	72	0 NO	1 NO	0 NO		0 NO	DISTENDED	NORMAL	LAP	NO	NONE		NORMAL		Multiple		0
Sapna	46	CHOLECYSTITIS	F	0	1	1	12 DAYS	NEGATIVE	DIABETES- 3 YEARS	0	PHC	90	0 NO	1 NO	0 NO		0 NO	DISTENDED	NORMAL	LAP	NO	NONE		NORMAL			1	0
Irfan	40	CHOLECYSTITIS	M	1	1	1	6 DAYS	NEGATIVE	NONE	0	PHC	78	0 NO	1 NO	0 NO		0 NO	DISTENDED	NORMAL	LAP	NO	NONE		NORMAL			1	0
Jawahar lal	56	CHOLECYSTITIS	M	0	1	1	12 DAYS	NEGATIVE	DIABETES- 2 YEARS	0	PHC	76	0 NO	1 NO	0 NO		0 NO	DISTENDED	NORMAL	LAP	NO	NONE		NORMAL		Multiple		0
Anita	34	CHOLECYSTITIS	F	1	1	1	15 DAYS	NEGATIVE	NONE	0	PHC	75	0 NO	1 NO	0 NO		0 NO	DISTENDED	NORMAL	LAP	NO	NONE		NORMAL			1	0
Gul Abha	30	CHOLECYSTITIS	F	1	1	1	9 DAYS	NEGATIVE	NONE	0	PHC	78	0 NO	1 NO	0 NO		0 NO	DISTENDED	NORMAL	LAP	NO	NONE		NORMAL		Multiple		0
Mrs Asha	40	CHOLECYSTITIS	F	1	1	1	1 DAY	NEGATIVE	NONE	0	PHC	80	0 YES	1 NO	0 NO		0 NO	DISTENDED	NORMAL	LAP	NO	NONE		NORMAL			1	0
Mrs Pushpa	38	CHOLECYSTITIS	F	0	1	1	5 DAYS	NEGATIVE	NONE	0	PHC	88	0 NO	1 NO	0 NO		0 NO	DISTENDED	NORMAL	LAP	NO	NONE		NORMAL			1	0
Amina	36	CHOLECYSTITIS	F	1	1	1	7 DAYS	NEGATIVE	NONE	0	PHC	80	0 NO	1 NO	0 NO		0 NO	DISTENDED	NORMAL	LAP	NO	NONE		NORMAL		Multiple		0
Mr reena	35	CHOLECYSTITIS	F	1	1	1	2 MONTHS	NEGATIVE	DIABETES- 3 YEARS	0	PHC	82	0 NO	1 NO	0 NO		0 NO	DISTENDED	NORMAL	LAP	NO	NONE		NORMAL		Multiple		0
Mamti Devi	45	CHOLELITHIASIS	F	1	1	1	6 MONTHS	NEGATIVE	DIABETES- 5 YEARS	0	PHC	2	0 YES	1 NO	0 NO		0 NO	NORMAL	NORMAL	LAP	NO	NONE		NORMAL		Multiple		0
Rekha	30	CHOLECYSTITIS	F	1	1	1	3 DAYS	NEGATIVE	NONE	0	PHC	72	0 YES	1 NO	0 NO		0 NO	NORMAL	NORMAL	LAP	NO	NONE		NORMAL		Multiple		0
Vivek	42	CHOLECYSTITIS	M	1	1	1	10 DAYS	NEGATIVE	DIABETES- 3 YEARS	0	PHC	74	0 YES	1 NO	0 NO		0 NO	DISTENDED	NORMAL	LAP	NO	NONE		NORMAL			1	0
Atul	41	CHOLECYSTITIS	M	1	1	1	14 DAYS	NEGATIVE	DIABETES- 3 YEARS	0	PHC	76	0 YES	1 NO	0 NO		0 NO	DISTENDED	NORMAL	LAP	NO	NONE		NORMAL			1	0
Indrita	43	CHOLECYSTITIS WITH	F	1	1	1	5 DAYS	NEGATIVE	DIABETES- 3 YEARS	0	PHC	78	0 YES	1 NO	0 NO		0 NO	DISTENDED	NORMAL	LAP	NO	NONE		NORMAL		Multiple		0
Dinesh	37	CHOLECYSTITIS	M	1	1	1	6 DAYS	NEGATIVE	DIABETES- 3 YEARS	0	PHC	70	0 YES	1 NO	0 NO		0 NO	NORMAL	NORMAL	LAP	NO	NONE		NORMAL		Multiple		0
Parvati	36	CHOLECYSTITIS	F	1	1	1	1 DAY	NEGATIVE	DIABETES- 3 YEARS	0	PHC	80	0 NO	1 NO	0 NO		0 NO	NORMAL	NORMAL	LAP	NO	NONE		NORMAL			1	0
Seema	31	CHOLECYSTITIS	F	1	1	1	20 DAYS	NEGATIVE	DIABETES- 2 YEARS	0	PHC	82	0 NO	1 NO	0 NO		0 NO	DISTENDED	NORMAL	LAP	NO	NONE		NORMAL		Multiple		0
Mukesh devi	60	CHOLECYSTITIS	F	1	1	1	1 MONTH	NEGATIVE	DIABETIC -20YRS	0	PHC	84	0 NO	1 NO	0 NO		0 NO	DISTENDED	NORMAL	LAP	NO	NONE		NORMAL			1	0
Sashi kumar	30	CHOLECYSTITIS	F	1	1	1	21 DAYS	NEGATIVE	NONE	0	PHC	86	0 NO	1 NO	0 NO		0 NO	DISTENDED	NORMAL	LAP	NO	NONE		NORMAL			1	0
Hanif Khan	41	CHOLECYSTITIS	M	1	1	1	5 DAYS	NEGATIVE	DIABETES- 3 YEARS	0	PHC	80	0 NO	1 NO	0 NO		0 NO	DISTENDED	NORMAL	LAP	NO	NONE		NORMAL		Multiple		0
Shakuntala	49	CHOLECYSTITIS	F	1	1	1	7 DAYS	NEGATIVE	DIABETES- 3 YEARS	0	PHC	68	0 NO	1 NO	0 NO		0 NO	DISTENDED	NORMAL	LAP	NO	NONE		NORMAL			1	0